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THE ANTIBODY TITRE IN MATERNAL AND INFANT'S SERUM AS AN INDICATION FOR TREATMENT IN HÆMOLYTIC DISEASE OF THE NEWBORN.

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SINCE the elucidation of the ætiology of hæmolytic disease of the newborn some ten years ago by Landsteiner and Wiener (1940) and by Levine *et alii* (1941), no worker has evolved an infallible method by which the severity of the disease in the infant can be forecast with certainty. Most are agreed that the clinical signs may be most misleading; an infant may appear normal at birth, be deeply jaundiced twelve hours later and be dead by the third day. Conversely, an affected, jaundiced infant at birth may survive with no apparent sequelæ.

One is obliged to look to the laboratory tests for the information which will clarify an otherwise obscure picture. Hitherto, even the best laboratory tests have given little real help. Curiously enough, attempts to evaluate the condition in the infant by measuring the "saline" antibody titre in another individual, the mother, have proved to be unreliable. Methods and techniques of performing titrations have been so numerous and diverse that there is little wonder that such scant information has been available from this source. Again, the three types of antibody, any of which may be present—whether the agglutinating form, the blocking form or the "third order" form—have all contributed to the confusion. Wiener *et alii* (1946, 1948), Bryce *et alii* (1951) and Allen *et alii* (1949) found a rough assessment possible by measuring the maternal antibody by the conglutination or albumin techniques, while Mollison and Cutbush (1949) found a good correlation between the levels of cord hæmoglobin and serum bilirubin when compared with the subsequent severity of the disease. They emphasize, and we would corroborate their findings, that the sample examined must be a cord sample of blood from the infant, since pronounced changes take place in the circulation after delivery. Dehydration, for example, may give an erroneously high hæmoglobin figure, which lulls the attendant into a sense of false security. The collection of cord blood samples is easy to arrange in a maternity hospital, but frequently no cord sample is available. Wherever possible the tests to be described later were performed upon cord blood samples, but we have found the tests to be reliable when carried out even two or three days after the birth of the infant.

Wiener *et alii* (1951) state that the antiglobulin test is a laborious procedure, though when carried out carefully it gives excellent results. After much trial and error we have adopted the antiglobulin test of Coombs *et alii* (1945, 1946) as a routine procedure. Both the direct and the indirect methods are employed and they have been standardized in every detail. Titrations are always performed by one person (G.V.) so that personal errors are minimized. The "saline" agglutinating test is most disappointing in the maternal serum, and like Wiener in one of his reported series (1948), we have not found a single instance of "saline" agglutinins in the serum of an infant in this investigation. The conglutination and albumin tests have been very variable in testing the mother's and the infant's serum and have seldom run parallel to the

antiglobulin test. The published results of titrations of mother's and infant's serum by Allen *et alii* (1949) and others employing the conglutination or albumin technique confirm the contention that such methods are, to say the least, erratic and fail to portray a picture in any way resembling that obtained by a standardized antiglobulin technique.

Technique of the Antiglobulin Test.

Indirect Method.

The indirect method of titration is as follows.

The specifically reactive test cells (usually group O CDe/CDe and O CDe/cDE cells) are washed three times in normal saline, then made up to a 10% solution in normal saline. The washing is a most important procedure and on no account may be omitted.

Place 0·4 millilitre of the undiluted serum to be tested in the first and second serial test tubes. In the second and each succeeding tube place 0·4 millilitre of normal saline. Then prepare serial dilutions in the usual manner by transferring 0·4 millilitre of the serum saline solution to each succeeding tube, starting from the second tube. Discard 0·4 millilitre from the final tube. To each tube add 0·4 millilitre of the above-mentioned 10% cell suspension. Then place the tubes in the water bath at 37° C. for one hour. (We have adopted the convention of calling the first tube "one in two" and not "full strength". Our titration results are therefore one dilution higher than they would be had we called the first tube "full strength" or "one in one".)

Wash the cells three times, using 10 volumes of normal saline solution, then remove the saline.

Add normal saline to two and a half times the volume of cells, thus giving a 40% cell suspension.

Place a drop of this cell suspension from each tube on a slide and add one drop of Coombs's reagent (Commonwealth Serum Laboratories) to each. The reagent is used according to the directions. We have found it to be specific, sensitive and uniform from batch to batch.

Gently tilt the slide from time to time and read the results in six minutes and not longer than ten minutes at room temperature.

Note at which dilution agglutination is just present as read by the naked eye. The reciprocal of the dilution is taken as the titre.

Rhesus-negative (group O cde/cde) control cells are used in parallel with the test cells.

Direct Method.

The direct method is as follows.

Five drops of the infant's cells are treated by being washed three times in five millilitres of normal saline, then made up to a 40% cell suspension in normal saline, to one drop of which one drop of Coombs's reagent is added. The results are read at once while the slide is being gently rocked at room temperature.

We have arbitrarily classified the results as "+++" when the agglutination is immediate and strong, "++" when the agglutination is delayed for up to two or three minutes and such large clumps are not formed, and "+" when the reaction is delayed up to five minutes or longer and the clumps of agglutinated cells are small and weak.

By employing the indirect test we have obtained titres up to 4096 in maternal serum and to 512 in infant's cord serum. We have found that a "+++" response to the direct test of the infant's cells corresponds with a high titre of antibody in the infant's serum (128 and higher), a "++" response with a titre between 8 and 128, and a "+" response with a serum titre of 8 or less.

Every case in this series has occurred in an Rh-negative (cde/cde or Cde/cde) mother whose serum has contained antibodies within the Rh system, being anti-D or anti-D + D^u, either alone or admixed with anti-C or anti-E. No other abnormal antibodies were detected in the serum of the mothers or infants concerned.

Results.

1. There have been six instances in which Rh-negative multiparous women have been mated to heterozygous men, and in whom previous immunization has occurred (Table I). Each of the six pregnancies under review resulted in the birth of an Rh-negative infant, while residual antibodies from previous immunization were still present. In every one of the six cases the titres of antibodies in the mother's and infant's cord serum were identical. The serum of one infant (Case IV) was examined at weekly intervals, and the antibodies were detectable for ninety-six days after delivery. This infant was breast-fed throughout the observations, but unfortunately the titrations of the milk were not followed. The hæmoglobin value of each child is within the normal range for a new-born infant. The direct antiglobulin test result was negative, as would be expected, in each case, and all infants survived. Jakobowicz and Bryce (1951) reported the appearance of an anti-M agglutinin in the serum of a mother and her newborn infant, which was of identical titre and persisted for five and a half months in the baby. Wiener *et alii* (1951) reported similar titres to ours in eleven cases of previously immunized Rh-negative mothers and their Rh-negative infants.

2. There were ten mild cases of hæmolytic disease of the newborn in the Rh-positive infants of Rh-negative mothers, in which the highest maternal titre was 64 and the highest infant's serum titre was 8. The direct antiglobulin test on the infant's cells in each case produced a "+" reaction according to the above-mentioned classification. The cases are set out in Table II. Three of the infants required simple blood transfusion only. In Cases VII and VIII the cord serum titre was 8, and the infants required simple blood transfusion on the fourteenth day. The infants in Cases XIIIA and XIIIB were identical twins, the maternal serum having a titre of 64, while both infants' sera had a titre of 4. Neither twin was jaundiced at any time, but one twin (Case XIIIA) was pallid with a falling hæmoglobin value; the child was given a simple transfusion of 100 millilitres of Rh-negative blood, but died on the following day. Post-mortem examination revealed bronchopneumonia. The surviving twin left the hospital

TABLE I.

Details of Six Rh-Negative Infants of Previously Immunized Mothers. The Mothers and Infant's Antibody Titres are Identical.

Initials.	Case Number.	Maternal Titre at Delivery.	Infant's Titre at Delivery.	Direct Antiglobulin Test Result.	Hæmoglobin Value. (Grammes *per Centum.*)	Birth Weight.	Weeks of Gestation at Delivery.	Remarks.
B.B.	I	128	128	—	18·0	lb. oz. 7 14	38	Rh-negative infant, not affected.
S.G.	II	128	128	—	17·4	5 0	36	Rh-negative infant, premature, slight jaundice on third day.
G.R.	III	64	64	—	17·6	8 14	39	Rh-negative infant, not affected.
W.D.	IV	64	64	—	17·4	8 3	38	Rh-negative infant, not affected.
T.N.	V	32	32	—	16·0	7 10	38	Rh-negative infant, not affected.
D.D.	VI	8	8	—	16·8	7 12	39	Rh-negative infant, not affected.

without receiving a blood transfusion, the hæmoglobin value on discharge being 12·8 grammes *per centum*. This infant received no treatment and served as a control for the infant which was lost. We feel justified, therefore, in holding that, although hæmolytic disease may have been a contributing factor, it was not the sole cause of the death of this child. In nine of the ten mild cases, therefore, the infants survived, with no treatment other than simple blood transfusion in the two cases in which the cord serum titre was as high as 8. With the exception of the infant who died and its twin, the hæmoglobin value was 14 grammes *per centum* or over in every infant. The twins were not examined by us until the third day, and the estimations were therefore not carried out on cord blood samples. In Case XIV labour was induced at thirty-eight weeks' gestation, because the mother had a previous history of neonatal deaths of her infants, while in Case XV labour was induced at thirty-nine weeks because of a rising maternal antibody titre.

3. There were 20 severe cases of hæmolytic disease, which are summarized in Table III. The maternal antibody titre at delivery varied from 64 to 2048 and the infant's cord blood serum titres varied from 16 to 512. In Case XVIII the mother, for religious reasons, refused to allow the infant to be given a blood transfusion. The infant served, therefore, as a control and received nothing but symptomatic treatment; the cord blood serum titre was 512 and the child died on the third day. Case XVII also served as a control; the infant was not given a blood transfusion and died when twenty-four hours old. This child was not selected as a control, but exchange transfusion was not carried out because of the absence of one of us. Case XXIX was a third control case, the cord blood antibody titre being 128. The infant was severely jaundiced and subsequently developed pronounced opisthotonus, tonic and clonic spasm and athetoid movement of classical kernicterus. A simple blood transfusion was given on the thirty-third day, and unfortunately the child survived as a mentally deficient infant for ten months. This infant (Case XXIX) was born on the same day as the infant in Case XXXIV, who died of tetany during the exchange transfusion, and was to have been given a transfusion immediately afterwards. As the cord blood hæmoglobin value was 15·4 grammes *per centum*, we felt justified on Mollison's (1949) criteria in leaving the infant without exchange transfusion, at the same time providing a control.

The remaining 17 severely affected infants were treated by exchange transfusion. The technique described by Wiener (1946) was employed, in which blood was given by the saphenous vein and removed from the radial artery. Certain modifications were made in some instances. One notable change has resulted from observations of tetany in three cases, and from the loss of one infant, who showed typical carpo-pedal spasm and accoucheur's hand.

We would take this opportunity to point out that the addition of sodium citrate to stored blood as an anti-coagulant immobilizes the ionizable calcium necessary for the clotting of blood, while at the same time basic sodium ions are added. Loutit *et alii* (1943) have pointed out that in the storage of blood, potassium ions are released from the cells into the plasma, so that the plasma potassium concentration rises with an increased length of time of storage. Our own estimations of plasma sodium and plasma potassium content on citrated blood stored for twenty-one days showed the plasma sodium content to be increased to 165 milliequivalents per litre, while the potassium content was increased to 30 milliequivalents per litre. The increase in basic sodium ions would produce a tendency towards metabolic alkalosis; this is perhaps not so important as the increase in potassium content, which at 30 milliequivalents per litre is highly toxic, particularly upon the heart muscle, causing well-known electrocardiographic changes and a tendency for the heart to stop in diastole. The action of the heart is also influenced by the blood calcium concentration, the potassium and calcium having a balancing effect upon one another (Best and Taylor, 1943). In utilizing stored citrated blood one is perfusing a fluid of high potassium and low calcium content, an unbalanced fluid, which would increase the length of diastole at the expense of systole, the heart finally coming to rest in diastole. It is our contention that in exchange transfusion with stored citrated blood cardiac overloading is in fact due to the unbalanced effect of potassium upon

TABLE II.

Mild Cases (Rh-positive Infants).

Initials.	Case Number.	Maternal Antibody Titre at Delivery.	Infant's Antibody Titre at Delivery.	Titre Difference.	Direct Antiglobulin Test Result.	Hæmoglobin Value. (Grammes *per Centum*.)	Birth Weight.	Simple Transfusion. (Days after Birth.)	Persistence of Infant's Antibodies. (Days.)	Infant's Hæmoglobin Value on Discharge from Hospital. (Grammes *per Centum*.)	Weeks of Gestation at Delivery.	Remarks.
L.O.	VII	64	8	56	+	14·5	lb. oz. 6 10	14		16·2	38	Slight jaundice on third day. Discharged on fifteenth day.
M. ..	VIII	64	8	56	+	16·4	4 11	14		13·4	37	Slight jaundice on second day. Discharged on nineteenth day.
B.G. ..	IX	64	8	56	+	16·6	7 0	—		12·4	39	Mild. Slight jaundice on third day. No transfusion.
H.T. ..	X	32	4	28	+	14·0	6 2	—	8	10·8	37	Mild. Slight jaundice on third day. No transfusion.
B.S. ..	XI	16	2	14	+	16·8	6 6	—	3	11·4	40	Mild. Slight jaundice on third day. No transfusion.
C.H. ..	XII	32	4	28	+	14·0	8 0	—	7	13·0	40	Mild. Slight jaundice on third day. No transfusion.
C.E.	XIIIA	64	4	60	+	8·4[1]	6 12	2			40	Pallor, no jaundice. Simple transfusion. Died (pneumonia).
C.E.	XIIIB	64	4	60	+	12·4[1]	6 10	—	9	12·8	40	Pallor, no jaundice. No transfusion.
R.T. ..	XIV	16	2	14	+	17·6	5 12	—	9	15·0	38	Not jaundiced. Very mild. No transfusion.
W.R...	XV	40	4	36	+	13·8	7 11	—	3		39	Mild. Jaundiced tinge on second day. No transfusion.

[1] Hæmoglobin value not estimated on cord blood.

TABLE III.

Severe Cases (Rh-positive Infants).

Patient's Initials.	Case Number.	Before Exchange Transfusion.						Amount of Exchange Transfusion. (Millilitres.)		After Exchange Transfusion.				Exchange Transfusion, Hours after Birth.	Citrated or Heparinized Blood.	Weeks of Gestation to Delivery.	Hæmoglobin Value on Discharge from Hospital. (Grammes *per Centum*.)	Death or Survival.	Remarks.
		Mother's Titre at Delivery.	Infant's Titre at Delivery.	Titre Difference.	Result of Direct Antiglobulin Test.	Cord Blood Hæmoglobin Value. (Grammes *per Centum*.)	Birth Weight	In.	Out.	Infant's Titre after Transfusion.	Amount at which Direct Antiglobulin Test Result became Negative.	Persistence of Infant's Antibodies. (Days.)	Reappearance of Reticulocytes. (Days.)						
L. ..	XVI	2048	512	1536	+++	14·0[1]	lb. oz. 7 4	700	700	64	Trace at 700			28	H.	40		D.	Death at 38 hours. Kernicterus. No autopsy.
G. ..	XVII	2048	128	1920	+++	11·4	5 14				Control					38		D.	Death at 24 hours.
C.M. ..	XVIII	2048	512	1536	+++	10·8	8 11				Control					40		D.	Death on third day.
R. ..	XIX	1024	128	896	+++	10·0	6 11	200	200					7	C.	37		D.	Exchange transfusion too small.
B.L. ..	XX	1024	256	768	+++	7·2	8 13	1000	800	16	300			3	H.	38		D.	Persistent occipito-posterior presentation. Autopsy: tentorial tear.
B.V. ..	XXI	1024	512	512	+++	8·0	5 11	410	430	128	Trace at 430	91	73	24	C.c.	39	8·4	S.	Three subsequent simple transfusions.
B.T.	XXII	1024	256	768	+++	11·0	6 5	800	800	16	400	57	28	4	H.	36	8·0	S.	
B.R. ..	XXIII	1024	512	512	+++	14·5[1]	5 15	500	375	256	+[2]	62+	62	6	C.c.	39	10·3	S.	Tetany. Revived with calcium gluconate. One simple transfusion.
S.F. ..	XXIV	1024	128	896	+++	9·8	6 14	345	250	32	Trace at 250	36	13		H.c.	38	14·5	S.	Two subsequent simple transfusions.
B.U. ..	XXV	1024	128	896	+++	15·0[1]	8 4	950	950	16	500	37	28	38	H.	40		S.	
A. ..	XXVI	512	64	448	++	11·6[1]	8 2	925	1000	4	400	20	5	10			8·6	S.	
B.W.	XXVII	512	64	448	++	17·2	7 12	890	890	16	400	20	13	10	H.	40	8·8	S.	
J. ..	XXVIII	512	32	480	++	17·0[1]	8 4	450	450	16	300	18	10		H.		10·3	S.	
C.L. ..	XXIX	512	128	384	+++	15·4	4 15				Control				C.	40		S.	Simple transfusion on thirty-third day. Mental defective. Death at 10 months.
S.C. ..	XXX	256	32	224	++	12·8	6 0	400	350	8	Trace at 350			24	C.	40	11·4	S.	
B.D. ..	XXXI	256	64	192	++	9·3[1]	6 2	450	400					44	C.	38	11·4	S.	
F.L.	XXXII	128	16	112	++	10·5	6 8	270	320					72	C.c.	38		S.	Tetany. One subsequent simple transfusion.
D.O. ..	XXXIII	128	16	112	++	11·4	6 0	400	350	4	250			24	C.	39	10·0	S.	
D.A. ..	XXXIV	128	32	96	++	17·6	7 4	375	360	16				72	C.			D.	Tetany. Death during operation.
I. ..	XXXV	64	16	48	++	13·6	8 0	410	470	Trace	380	2	26	4	H.	41	11·4	S.	

[1] Hæmoglobin value not estimated on cord blood. [2] Remained positive for nineteen days.

the heart. This becomes especially true when large volumes of blood are transfused and removed. It is conceivable that such an effect would also occur in adults with large volumes of transfused blood, particularly if the plasma potassium level was already raised. We have, therefore, discarded the use of citrated stored blood for exchange transfusion purposes and have substituted heparinized whole blood which has been freshly collected into waxed bottles. Two of the infants were fortunately revived by the intravenous administration of calcium gluconate when we were using blood collected into acid citrate dextrose solution (Medical Research Council solution number 2).

We have not encountered any apparent tendency to cardiac overloading, since fresh heparinized blood has been substituted for citrated blood. The troublesome cyanosis, profuse salivation and irritability so often found when citrated blood is used no longer occur with heparin. In fact, the infant usually goes to sleep during the operation. In the use of heparinized blood any untoward bleeding tendency is controlled by protamine sulphate, which is injected intramuscularly (a 1% aqueous solution in equivalent quantity to the number of millilitres of heparin—1000 international units per millilitre—which have been given in the transfused blood).

Nevertheless, in order to avoid the possibility of overloading the cardio-vascular system we have endeavoured to maintain the infant in a negative blood balance during the operation of exchange transfusion. That is to say that slightly more blood is removed from the circulation than is going in.

In an attempt to discover the efficiency of exchange transfusion various investigations were made. At intervals during the operation samples of the outcoming blood were collected into dry, heparinized tubes for laboratory examination. Such blood consists of a mixture of Rh-positive and Rh-negative blood, the negative fraction increasing at the expense of the positive fraction as the operation progresses. The disappearance of the positive direct antiglobulin test result would therefore tell us roughly when all of the infant's own affected Rh-positive cells had been removed. Previously, serial mixtures of washed Rh-positive and Rh-negative cells had been made by mixing 49 parts of Rh-positive to one part of Rh-negative cells, 48 parts of Rh-positive to two parts of Rh-negative cells, and so on to one part of Rh-positive cells to 49 parts of Rh-negative cells. The mixtures were then subjected to the indirect antiglobulin test. It was found that approximately one part of Rh-positive cells with 49 parts of Rh-negative cells could not be detected by the naked eye, although an occasional clump could be seen, microscopically, and that there could not therefore be more than 2% of detectable Rh-positive cells in the mixture. It was surprising that we found that the affected Rh-positive cells could be removed from the baby by the withdrawal of small quantities of blood. In only one case was as much as 500 millilitres of outcoming blood required (Case XXV); in other cases the affected cells were removed by as little as 250 to 450 millilitres of blood, depending upon the weight of the infant. In the subsequent three or four days the direct antiglobulin test result remained negative; this rules out the possibility that depot cells were being released into the circulation in detectable quantities. As will be explained below, the direct antiglobulin test result became transiently positive several weeks later in some cases.

Although it was comparatively easy to remove the infant's affected cells, we found that it was extremely difficult to remove the antibodies from the child's serum by exchange transfusion. In one infant whose initial titre was low (Case XXXV) they could be almost removed, but in those with a high initial titre, 950 millilitres of exchanged blood reduced the titre from 128 to 16 (Case XXV). In Case XXI, although the affected cells were removed by 430 millilitres of exchanged blood, the antibody titre was reduced from 512 to 128 only, whereas in Case XXII 800 millilitres of blood reduced a titre of 256 to 16. A high titre of residual antibodies after exchange transfusion appeared to depress hæmatopolesis; one infant (Case XXI) required three subsequent simple blood transfusions, whereas another (Case XXII) required no further blood when the titre of residual antibodies was low after the operation. The infant in Case XXI required seventy-three days before reticulocytes reappeared in the circulation despite a falling hæmoglobin value; the infant in Case XXII required only twenty-eight days with similar hæmoglobin behaviour. Upon the reappearance of numerous reticulocytes the direct antiglobulin test result again became positive for a few days, then the antibodies disappeared from the serum and the hæmoglobin value finally commenced to rise (Case XXII, Figure I).

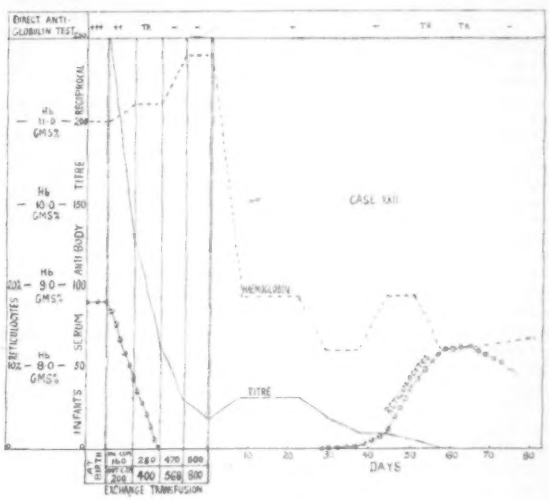

FIGURE I.

Showing behaviour of infant's serum antibody titre, result of direct antiglobulin test, hæmoglobin value and reticulocyte count during exchange transfusion and subsequently.

It is thus clear that antibodies remaining in the infant's circulation to any pronounced degree will continue to cause erythrocyte destruction until they finally disappear. In any case they spontaneously disappear, as was shown in the Rh-negative infant in Case IV, in which an initial titre of 64 took ninety-six days to disappear. It seems, therefore, to be of little value to prescribe iron and liver extract for these affected infants until the antibodies have finally disappeared from the serum and reticulocytes have reappeared. This emphasizes also the importance of being quite sure that the Rh-negative blood which is transfused into the infant contains no antibodies. Although some of the blood used in this series was from parous women, none contained detectable antibodies. In contrast to Allen *et alii* (1949), we would prefer male donor blood, for the reason that it is less likely to contain antibodies if antibody tests of the donor's blood had been omitted. Male and female donor blood has been transfused at random in this investigation and appears to have made little difference to the results.

The infant in Case XVI was desperately ill, severely jaundiced and almost moribund when first examined twenty-four hours after delivery. As an experiment we felt justified in giving both Rh-positive and Rh-negative blood in an attempt to absorb and remove the circulating antibodies. One hundred millilitres of Rh-negative blood were given first and the radial artery was opened while this was running in; then 200 millilitres of Rh-positive blood were given, and finally 400 millilitres of Rh-negative blood were given. The last sample collected before the radial artery was tied at the end of the operation showed a trace only when subjected to the direct antiglobulin test. The same sample showed antibodies to be still present to a titre of 64.

Since the giving of Rh-positive cells failed to reduce the antibody titre sufficiently, we think that the experiment was not successful; but we feel justified in taking the risk because Rh-positive blood used to be given by transfusion to babies suffering from hæmolytic disease of the newborn

before the discovery of the Rh factor, and many such patients survived after this old treatment.

This infant (Case XVI) had been subjected to the full force of the powerful antibodies *in utero* until the fortieth week. Since the maternal antibodies (Watson, 1951) may build up rapidly to a high titre in the last four weeks of gestation, we still advocate the induction of labour after the thirty-sixth week if the maternal antibody titre is high or rising. Two of the control infants (Cases XVIII and XXIX) were delivered at forty weeks, and one (Case XVII) at thirty-eight weeks' gestation.

In Case XX there had been a persistent occipito-posterior presentation. At delivery there was no sign of jaundice; the infant was pale and the breathing was spasmodic and irregular. Although it was not recognized at the time, the differential diagnosis lay between *asphyxia pallida* and anæmia due to hæmolytic disease of the newborn. When the laboratory report was given of a cord serum antibody titre of 256 and cord blood hæmoglobin value 7·2 grammes *per centum*, it was decided to perform exchange transfusion, which was commenced three hours after the infant's birth. Heparinized donor blood was used and the exchange was performed in balanced volumes of ingoing and outcoming blood. When 800 millilitres had been removed the radial artery was tied and a further volume of 200 millilitres of blood was given; but the infant remained pallid even after this extra volume had been given. The transfusion was continued after 800 millilitres because at 850, 900 and 950 millilitres no amelioration of the pallor occurred.

Autopsy revealed a tentorial tear, but apparently the injection of protamine sulphate had controlled the hæmorrhage, since only approximately two to three millilitres of old blood could be detected in the adjacent structures, much of it being clotted. However, we believe that there is some risk in using heparinized blood in cases in which cerebral damage may have occurred. Furthermore, while we fully appreciate the risk of over-transfusion, it is apparent that in this case an excess 200 millilitres may have contributed to the fatal outcome.

4. There were seven still-born infants in the series. The maternal antibody titres ranged from 4096 to 1024. Fœtal deaths occurred between the twenty-sixth and the thirty-ninth weeks. The results are tabulated in Table IV. In

TABLE IV.

Details of Seven Stillborn Rh-positive Infants, Maternal Titre and Weeks' Gestation at Death of Infant.

Initials.	Case Number.	Maternal Titre at Delivery.	Weeks' Gestation at which Infant Died.
M.L. ..	XXXVI	4096	38
G.N. ..	XXXVII	4096	26
M.N. ..	XXXVIII	1024	32
T.N. ..	XXXIX	1024	28
B.W. ..	XL	1024	32
L.Z. ..	XLI	1024	39
S.D.	XLII	2048	32

only one case (Case XXXVI) was the serum of the infant in a sufficient state of preservation for titrations to be performed. In this instance the fœtal heart sounds ceased approximately three days before delivery at the thirty-eighth week of gestation. Blood was collected from the heart of the infant soon after delivery and at once subjected to the laboratory tests. The direct antiglobulin test applied to the red cells produced a "+++" reaction, the serum titre being 64 when estimated by the indirect method.

Discussion.

The finding of an equal antibody titre in the serum of the previously immunized mothers and their Rh-negative infants shows that there was no barrier to the transfer of antibodies from mother to infant. On the other hand, when the infant was Rh-positive, the antibody titre of its serum was always at least one dilution less than that of the mother. This would at once suggest that the antibody had been absorbed by the infant and that the degree of absorption could be measured by the difference between the mother's and the infant's titres. Thus a high maternal titre of 1024 and an infant's titre of 128 would suggest a very pronounced absorption resulting in a badly affected infant—that is, an absorption of 896 units of antibody by the child. That the antibody is absorbed upon the child's red cells is evidenced by the result of the direct antiglobulin test; the degree to which it is absorbed is reflected in the degree of the reaction to this test.

Bearing in mind the gross inaccuracies of the doubling dilution method of titration, when we come to examine the titre differences in this series we find that in the case in which the titre difference was 4032 units the infant was stillborn (Case XXXVI); when the difference was 1920 units and the child was untreated (Case XVII) it died in twenty-four hours; in another case in which the difference was 1536 units the child died on the third day; while another control with a difference of 384 units survived temporarily as a mentally defective child. With the exception of the infant who was lost as a result of tetany (Case XXXIV), all the deaths occurred in infants with a titre difference greater than 750 (Cases XVI, XVII, XVIII, XIX and XX). When exchange transfusion therapy was given, three infants with titre differences between 750 and 1000 survived (Cases XXII, XXIV and XXV), as also did all those with a titre difference as low as 48. Simple transfusion only appeared to be necessary when titre differences were below 50, but some infants survived even without this (Case XIIIB). A "+++" response to the direct antiglobulin test corresponds roughly with a titre difference of approximately 500 units or more in every case except Case XXIX. A "++" reaction corresponds with a titre difference between 50 and 500, while a "+" reaction corresponds with a titre difference between 2 and 50 units.

As has already been mentioned, although affected red cells could be removed effectively by adequate exchange transfusion, in no instance were we able to remove all the antibodies from the serum of an infant. In fact, in the subsequent examinations of the blood in Case XXII (Figure I), the antibody titre rose from 16 to 32 in the three weeks following the operation, and then slowly disappeared. This phenomenon has been observed in many other similar instances.

Veall and Mollison (1950) have pointed out that if an infant is anæmic before exchange transfusion, a much smaller volume of blood will be required at the operation to remove all the affected cells than has been calculated by Wiener and Wexler (1946). Wiener has calculated that during simultaneous exchange transfusion the change of one equivalent volume of the infant's blood leaves 63·2% of donor blood in the infant, that of twice the infant's blood volume leaves 86·5%, and that of three times the infant's blood volume leaves 95% of the donor's blood in the infant.

Our finding would suggest that both opinions are largely correct, according to whether the red cells alone or the whole blood is being considered, seeing that we have "tagged" both the erythrocytes and the plasma by the direct and indirect antiglobulin tests respectively. If exchange transfusion is efficiently performed with whole heparinized blood, instead of with diluted citrated blood, the final addition of concentrated red cells should not be necessary in order to restore a normal red cell population. However, as Mollison points out, the transfused cells will themselves be normally destroyed at the rate of 1% per day, and if hæmatopoiesis is temporarily suspended while antibodies are still present in the infant's plasma, a falling hæmoglobin level is inevitable even after exchange transfusion.

It is clear that exchange transfusion does in fact remove the affected cells and lower the antibody concentration in the infant's serum to safe levels; the lower the initial titre, the easier it is to bring it to a satisfactory figure (Cases XXX, XXXIII and XXXV). The higher the initial titre,

the more blood is required to be exchanged to bring about the same result (Cases XXII, XXV, XXVI and XXVII), and if insufficient blood is used at exchange transfusion, subsequent simple transfusions will be required (Cases XXI, XXIII and XXXII).

We have endeavoured to reduce the infant's serum titre to 16 or lower; this we have taken as the critical level for a satisfactory operation. The volume of blood required, as suggested by Wiener (1946), is calculated as being three times the infant's blood volume. This in turn is taken as 40 millilitres per pound of body weight. In Case XXI 690 millilitres should have been given by this calculation, whereas only 430 millilitres were given, and this is less than twice its estimated blood volume. The sequel was a series of simple blood transfusions.

Case XXXV was the only one in which the antibodies were almost entirely removed. The infant's initial titre was low; the titre difference was 48, and although the child weighed eight pounds, only 470 millilitres of blood were removed. Sixty millilitres more blood were removed than was actually given, and in spite of this no further simple transfusion was required; the child's hæmoglobin value on discharge from hospital was 11·4 grammes *per centum*. This was a borderline case in which the child would probably have done well by simple transfusion alone; but we believe that the exchange transfusion performed four hours after birth prevented the appearance of jaundice, saved the mother and attendants a great deal of worry and allowed the mother to take home a normal infant on her departure from hospital.

It is our belief that infants affected with hæmolytic disease do not die of anæmia *per se*. They die as a result of the antigen-antibody reaction effect upon the tissues, whether this is due, as Wiener contends, to clumps of agglutinated cells causing emboli and anoxæmia, or whether the antigen-antibody effect takes place on the tissue cells themselves. It is indeed surprising that the fœtus can withstand for forty weeks the effects of an incompatible environment and yet survive. Its tissues and red cells can resist for months the attacks of hostile antibodies, which in an adult would cause a severe reaction in a matter of minutes. *In utero* the absorption of antibodies upon the cells of the infant appears to be a very slow process, while the subsequent destruction of cells is even slower. The time during which the process of absorption is permitted to continue is of importance. If the maternal antibody concentration were to remain constant, it is suggested that the infant's serum antibody titre would fall progressively as absorption occurred upon its cells. Thus in Case XIX, in which the maternal titre was 1024, the infant's titre was 128 and the titre difference was 896, the child was far more severely affected (and died) than in Case XXIII, in which the maternal titre was also 1024, but the infant's titre was 512, the titre difference being 512. In the case of the stillborn infant (Case XXXVI) the mother's antibodies to a very high titre had been almost entirely absorbed by the infant's cells, since only 64 units still remained free in the child's serum.

In Case XIV, a very mild case with a "titre difference" of 14, the infant was delivered at thirty-eight weeks' gestation. The indirect antiglobulin titration on the cord serum gave a value of 2, while the direct antiglobulin test gave a "+" result, the hæmoglobin value being 17·6 grammes *per centum*. Only eight days later the indirect antiglobulin test on the serum gave a negative result, while the direct test now produced only a "trace reaction". Two days later both the indirect and direct tests gave negative results, while the hæmoglobin value was now 15·0 grammes *per centum*. In this mildly affected infant, therefore, all traces of antibodies had disappeared spontaneously in nine days after delivery, while no transfusion therapy had been instituted. However, the antibodies in this time appear to have taken some toll of the red cells, since the hæmoglobin value had fallen from an initial figure of 17·6 grammes to 15·0 grammes *per centum*. Not more than the merest trace of jaundice had been manifest during this time. The reticulocyte percentage was 0·3 on the ninth day.

Both Case XIV and Case XXII are given as typical examples of the antibody behaviour in mildly and severely affected infants, with no treatment and exchange transfusion respectively.

None of the babies in this series, except the Rh-negative infants, have been breast-fed by the mother, the main reason being that we wished to study the antibody titre in the infant after exchange transfusion. If, as we still think it possible in the very young infant, the antibodies from the milk had been absorbed from the gut of the child, the results would have been vitiated.

It seems apparent that the severity of the disease can be forecast to some degree by the antibody titre in the mother's serum at the time of delivery. An even better forecast is obtained when the antibody titre in the infant's blood is obtained and the difference between the maternal and infant's titres is estimated. The degree of the reaction to the direct antiglobulin test of the infant's cells gives confirmatory evidence. If the maternal titre is very high (4096) the infant will probably be stillborn; if it is high (1024 to 2048) the infant will probably be stillborn if allowed to go to term, or it may be born alive and die soon after birth unless drastic treatment is instituted. It may die even in spite of drastic treatment (Case XVI). We have two instances in which the maternal titre was 1024, the infants' cord serum titre was 512 and the infants survived (Cases XXI and XXIII) after small exchange transfusions, additional simple transfusions being given subsequently.

In the low titre cases, in those infants studied the persistence of antibodies in the serum was of short duration only (Cases X, XI, XII, XIIIB and XV), and the infants made uninterrupted recoveries. Exchange transfusions in the severe cases brought the titre down to lower levels; but in spite of this, the persistence of antibodies ranged from two to ninety-one days, depending upon the infant's initial cord blood titre. On these grounds we strongly advocate the termination of pregnancy if gestation has progressed beyond thirty-six weeks and the maternal titre is high or rising. One must balance the hazards of prematurity against those of allowing the infant to continue to term in an environment of potent antibodies. The time to induce labour is decided by consultation with the obstetrician, and the best method of delivery is *per vaginam*. We do not advocate Cæsarean section, which only superadds a risk upon the mother and does not enhance the chances of the infant's survival.

The purpose of this work has been to attempt to clear up in our minds the problems which used to arise when an infant suffering from hæmolytic disease of the newborn was found. Although we have practised exchange transfusion in this hospital since 1945, we had never been quite sure of the indications. For some time before the foregoing standardized titration technique had been evolved, infants whose mothers had incompatible antibodies and whose cells yielded a positive response to the direct antiglobulin test were given exchange transfusion. Most of these recovered, but as many of these transfusions were probably unnecessary, publication of the results of these early cases would be worthless. On the other hand, when infants were left untreated or were given only simple transfusion, a number died.

Not only in this series, but among patients previously treated, we have not seen a case of mental defectiveness in a surviving infant who had been adequately treated by exchange transfusion; however, we have seen this occur in babies who were either left untreated or treated by simple transfusion alone.

The answer to the problem appears to be that some infants require exchange transfusion, some simple transfusions and some no treatment at all, each case being judged on its individual merits, as determined by titration.

The suggested antenatal examinations with the interpretation of the results is as follows.

1. An early antenatal check of the mother for Rh status should be made. If the mother is Rh-negative, the father should be tested for his genotype. When necessary, any siblings should be Rh typed.

2. When applicable, a periodical monthly examination for antibodies in the mother should be carried out. (*a*) If antibodies are present at any time the frequency of testing is altered to once a week after the thirty-second week. (*b*) If the titre of antibodies is above 64 and rising, termination of pregnancy is advised at the thirty-sixth week. (*c*) If the father is homozygous and the maternal titre high and stationary, we advise termination of pregnancy at thirty-six weeks. If the father is heterozygous and the titre is stationary at a high level, we would still advise termination of pregnancy; if it is stationary at a low level, we would allow the pregnancy to proceed and watch the titre at weekly intervals, since the infants of heterozygous fathers may be Rh-negative. In this series a dangerously high maternal titre was found only when the father was homozygous.

Conclusions.

A standardized direct and indirect antiglobulin test employed in this series of 43 cases, which have occurred in this hospital in the last eighteen months, has provided us with an instrument which, we believe, can accurately forecast the severity of the disease to be expected in hæmolytic disease of the newborn. We have found the tests to be extremely sensitive when handled according to the description given in the text. Furthermore, the results of the tests have been uniformly consistent, whereas the conglutination and albumin titrations have been erratic and misleading even when performed under standardized conditions.

By the use of the serum from the maternal blood collected at delivery and of the cord blood from the infant, the antiglobulin tests have clarified the detection and measurement of the antibodies acquired by the infant *in utero*. The subtraction of the infant's serum titre from that of the mother gives a measure of the antibodies absorbed by the cells of the child and is referred to as the "titre difference". Whether this absorption is upon the erythrocytes alone or upon the tissue cells in addition, has still to be definitely proved.

Exchange transfusion, which has been widely adopted as a method of treatment both in England and in America, has few protagonists in Australia. That it can be a life-saving measure in severely affected infants cannot be doubted. When performed efficiently it not only removes the affected red cells, but also lowers the antibody titre in the serum, the degree of lowering depending upon the amount of blood exchanged. The empirical exhibition of liver and iron therapy to an anæmic infant in whom incompatible antibodies are still present has little logic, and the continued presence of such antibodies readily explains the necessity for repeated small simple transfusions. Clinically, the improvement in an infant severely affected and treated efficiently by exchange transfusion is dramatic when compared with the condition of a similarly affected infant treated by simple transfusion or given no treatment at all.

It should be appreciated that we do not advocate exchange transfusion in all cases. In our series we have noted the hazards of using citrated blood and have substituted the use of heparinized blood.

The laboratory tests described enable the clinician to classify his patients into those requiring exchange transfusion, simple transfusion or no treatment at all.

Summary.

1. Standardized antiglobulin tests are described for the determination of antibodies in mothers and infants affected with hæmolytic disease of the newborn.

2. (*a*) The titration of the maternal antibodies at delivery gives an indication of the prognosis for the infant and of the treatment to be adopted. (*b*) The titration of the antibodies and the reaction of the red cells in the infant's cord blood give an even better indication of the severity of the disease to be expected in the infant. (*c*) The significance of the difference in antibody titre between the mother's and infant's serum is suggested. (*d*) A series of 43 cases is reviewed.

3. The behaviour of antibodies in the infant's serum after delivery is shown.

4. The rationale of exchange transfusion is defended and erythrocyte and serum antibody changes occurring during this operation are demonstrated.

5. An antenatal routine for the testing of the parties concerned in anticipated hæmolytic disease of the newborn is suggested. An interpretation of the laboratory findings is presented.

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THE ENURESIS DYAD.

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A CONCEPT that the ætiology of enuresis is connected with frustration occurring during early toilet training arose from an investigation of 73 mothers whose children attended a kindergarten. Forty-three children had been rigidly trained; 26 were enuretic. Of the remainder, trained without coercion, three only were enuretic (Bostock and Shackleton, 1951).

Corroborative evidence of this relationship has now been obtained from close analysis of 73 case histories of children attending the Child Guidance Clinic at the Brisbane Children's Hospital. This forms the basis of the present communication.

Interrogation of the mothers concerning toilet training was made by one of us (M.G.S.), the same technique as in the previous research being used.

The cases were not chosen solely on the score of enuresis. Many enuretics were, in fact, left out of the series because we did not consider that our data were complete.

The results were epitomized in Table I.

TABLE I.

Condition.	Rigid Training of 38 Children.	Training Without Coercion of 35 Children.	Total.
Enuretic	34	2	36
Non-enuretic	4	33	37
Total	38	35	73

The figures strongly support our previous conclusion. The incidence of enuresis is considerably higher among children who have had rigid toilet training.

It would seem that there is an "enuresis dyad". This may be set out as follows: rigid training, frustration and possibly fear create a habit of continence and an initial "dry bed"; but later frustration and possibly fear produce regression and stereotype, with a subsequent "wet bed". The foregoing confirms the contention of Maier (1949) that enuresis is a stereotypy, unmotivated, unadaptive and without goal. It is regressive, aimless behaviour initiated by frustration.

Therapy.

If frustration plays such a large part in the genesis of enuresis, it would seem reasonable to suppose that psychological treatment on the lines of its reduction may produce good results.

Space will not permit presentation of the entire case records of our patients. Improvement was achieved in 16 cases. This is a low estimate, as in the changing clientele of an out-patient clinic it is difficult to be precise. The cases must be viewed against a background of chronicity. Almost without exception our patients had previously received orthodox treatment on the lines of sedation, fluid restriction, diet or stimulation. They had been cajoled, threatened, punished and praised in conformance with current views that enuresis is a motivated activity. Under the circumstances the results are in accord with the dyad concept, since in the management of a guidance clinic, which can never completely control the home conditions, the entire removal of the frustration factor is probably an impossibility.

The following six cases were selected at random. They illustrate the *modus operandi* of investigation and therapy. After full investigation of the total situation, steps are taken to alter the environment and to change the child's attitudes towards its surroundings.

CASE I.—Jessie, aged ten years, is the youngest of three children. Her brothers are aged eighteen and twenty-two years. Jessie has wet the bed since she was three and a half years old. Dryness was achieved early—at about fifteen months. The toilet training, begun early, was pursued with unrelenting regularity and tenacity. Scolding and slapping were the punishments for failure. The mother related that she had found the little girl very trying to train in habits of tidiness and neatness; however, she had persevered, and the child developed a shrinking sensitivity, irritability, and later enuresis.

The mother is a house-proud, obsessively tidy and methodical woman, who strictly supervises her daughter's every activity. She endeavours to instil quiet habits, such as doing needlework, instead of childish play. The girl has a long list of household duties to perform, both before and after school.

The rationale of the problem was described to the mother, but very little progress was made in changing the attitudes of a lifetime until the mother was forced to stay in hospital for some months. The chance was then seized to place Jessie in charge of the household, and to allow her initiative in organizing her own life. She was given scope to develop a feeling of being necessary to her father and brothers.

To Jessie's great satisfaction her bed-wetting cleared up before her mother returned from hospital. The new regime was too well established to be easily upset and the cure has been maintained for over twelve months.

CASE II.—Gladys, aged three years, was brought to us because she had relapsed into bed-wetting after having had a dry bed for eighteen months. She had a brother aged sixteen months.

Toilet training had been very early and rigid. The mother desired to produce a perfect child in the mould of those she imagined were portrayed in books on child guidance. Her half-understood theories were thrust on Gladys. Great was her chagrin when the child began to wet the bed shortly after the arrival of a brother. The child very quickly showed that she resented the newcomer, and would attack the baby whenever possible. The mother reacted to this with dislike and anger verging on cruelty.

After many sessions of trying to enlighten the mother concerning the basis of her daughter's frustration, opportunity arose to place the child in a toddlers' home for six months. Here she received affection and attention of which she had hitherto been deprived. At the end of six months she returned to her mother. Her bed-wetting had disappeared and many of her other behaviour disabilities had been ameliorated. Fortunately it was possible for her to attend a kindergarten near her home, and to this date there has been no recurrence of the enuresis. In the meantime her mother had gained some insight and was noticeably kinder to the child.

In the above-mentioned cases a cure was effected despite the handicap provided by mothers who at first seemed incapable of gaining insight into the problem. Manipulation of environment gave the children a chance of normal development free from frustration; improvement was maintained in Case I because Jessie's maturity presented the mother with a *fait accompli* on her return, and in Case II the environment was bettered by the aid of the daily sojourn at the kindergarten and the mother's ultimate change of view.

CASE III.—Winnifred, aged five years, was brought to us by her mother, who was greatly distressed at the child's continued hostility to herself and the stepfather. Six months had elapsed since remarriage after divorce. She now proposed taking the child to live with her and her new husband. The child strenuously objected to the arrangement and became uncontrollable in the mother's presence. She insisted upon remaining with her grandmother. Enuresis appeared at this time and was a nightly occurrence.

The case history revealed a record of great marital unhappiness during the first marriage, with intense resentment of pregnancy. Early and rigid toilet training had

produced the initial "dry bed". The mother admitted that in her unhappy state she often was extremely "cross" and "took it out" on baby. After the divorce the child saw her father once a week until she was brought to us.

Projection technique disclosed a disturbing Œdipus situation. The accompanying picture (Figure I) is revealing. It depicts a wedding staged by the child, in which she is married to her own father. Her mother, whom she had identified with one of the dolls, was thown out of sight. The stepfather was buried in a closed suitcase; the child subsequently sat on it.

FIGURE I.

On a second occasion she portrayed her real father's wedding to her mother; she was present as "flower-girl"; the stepfather remained in the suitcase. Figure II shows this.

A subsequent visit by the social worker revealed cessation of the enuresis. The child had returned to her grandparents and thereby dodged her problems.

FIGURE II.

This case reveals a clear-cut picture of an initial "dry bed" and a subsequent "wet bed" based on a process of continued frustration.

CASE IV.—Mabel, aged ten years, was brought to us because of backwardness at school. She was working in a class with children three years her junior and failed to hold her own even at this lowered standard. Enuresis was not reported until a subsequent interview, when it was disclosed that Mabel had wet the bed nightly since she was aged six years. The intelligence quotient was 109. Inquiry elicited the following facts: Mabel is the eldest of three girls. When she was aged six years her mother went to hospital for the birth of a stillborn son. She returned a tired and weary invalid. Our patient was called upon to do most of the household duties. Subsequently the dead baby brother was idealized, and though living only in the spirit world he took a beloved place in the family circle. Later, another girl was born, and Mabel's life of duties became even busier.

The extraordinary situation of a living dead brother was portrayed in an interview during which Mabel played with "Plasticine". She created a domestic situation in which everyone was present except herself. To the various family

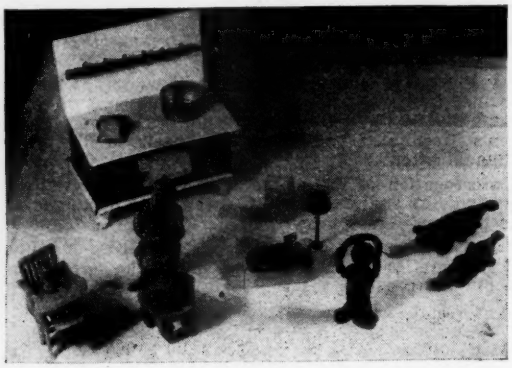

FIGURE III.

figures she assigned ages corresponding to their own. The dead child was modelled and placed on a couch with a book of comics in his hand. She regarded him as a four-year-old. Figure III illustrates this scene.

Mabel's toilet training had been early and rigid. She achieved the initial "dry bed"; the subsequent "wet bed" had commenced shortly after her mother returned from hospital after giving birth to the stillborn son. The habit persisted.

Treatment commenced by explanation to the parents of the dire consequences of Mabel's feeling of rejection. Cooperation was excellent. They persuaded the child that she was loved and wanted for herself alone. The dead brother was

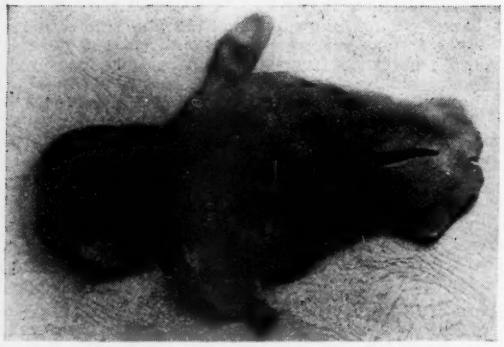

FIGURE IV.

banished as a topic of conversation. Mabel was released from her never-ending chores.

These procedures wrought a minor miracle. Mabel's appearance changed from one of lifelessness and apathy to one of alertness and fun. She made strides in overtaking her lag in school work and was quickly promoted to a higher grade. The bed-wetting ceased.

CASE V.—Henri is a "New Australian", five and a half years old, with an intelligence on the Merrill-Palmer scale rated at "120+". His mother consulted us for his inability to learn English. This was in startling contrast to his achievement of fluency in two other languages. The problem of enuresis was mentioned incidentally.

The case history disclosed maternal reluctance towards parenthood and an intense drive to achieve a perfect child. His behaviour became a matter of parental self-glorification or of reproach. The toilet training had been impatient, regular and unremitting. Punishment accompanied all failures. Henri had not even achieved the initial "dry bed".

A sister was born when Henri was three and a half years old. The father made no secret of his preference for her.

The condition yielded to treatment on the lines found successful with other bed-wetters, though in this case the problem of securing the parents' cooperation was unusually difficult. They were reticent and felt they were strangers in a strange land. Since they were intensely eager to make good in a new country, ordinary difficulties assumed astronomical proportions. Their son's failures were therefore much harder to condone. Fortunately, close liaison was maintained with them through the social worker of the Immigration Department. Both parents did their best to cooperate. The mother ignored the child's bed-wetting and ceased to urge him to excel at school; the father showed less preference for his daughter and spent more time with his son.

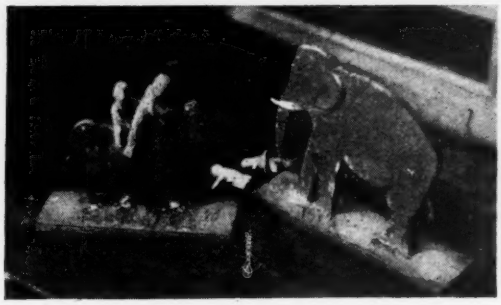

FIGURE V.

The results were gratifying. Henri's teacher at school reported substantial progress. Interest took the place of aggression. The enuresis disappeared.

CASE VI.—Joan is eleven years old. Her brother, aged six years, is the veritable "apple of his mother's eye". The mother is both elderly and irritable. She reported that, after a toilet training in which an initial "dry bed" was achieved at twelve months by dint of smackings and scoldings, there was the subsequent "wet bed" three months after the birth of a brother. It has continued ever since.

The child soon revealed that she regarded her brother as the bane of her existence and thought she herself was of no account in the family. Figure IV shows her brother modelled in "Plasticine", rolled flat with a bottle as though under a steamroller.

This clear-cut case of sibling rivalry, with its attendant frustration, has produced a defiant, aggressive child who managed to make her ineffectual mother's life completely miserable. The slow work of reorientating the mother towards her daughter, and of persuading the father to assist in the process, involved some eight months. At the end of that period Joan regained the initial "dry bed" every night, and her other conduct disorders also decreased.

In the above cases factors involved were invariably early and rigid toilet training, followed in most cases by an initial "dry bed". Then came later insecurity, in the shape of sibling rivalry, maternal rejection, parental restrictions on the healthy development of a growing child, or some other factor which produces a feeling of frustration, resulting in the subsequent "wet bed".

It must be stressed that frustration alone will not produce the enuresis dyad. Enuresis is usually an unfortunate and distressing side-effect of early and rigid toilet training. It is a stereotypy based on a previous frustration.

We believe that enuresis is seldom used as a means of obtaining an objective. It has been stated that enuresis often solves a motive of revenge, or achieves a feeling of self-glorification because of the attention which ensues. Such beliefs are untenable in view of the evidence. This is illustrated in the following section.

Frustration Without the Enuresis Dyad.

Since enuresis occurred in only two out of 35 children in this series who have been toilet trained without coercion, it seems unlikely that later frustration *per se* is a major factor. The frustrations to which these children were subjected were entirely comparable with those already quoted. The following five cases without enuresis illustrate this.

CASE VII.—Tom, aged eight years, was so anti-social that his head teacher asked his parents to remove him from the school. He threw stones at other children and at school windows, was defiant to teachers and cruel to his playmates; he was disobedient at home, aggressive and irritable, and given to bouts of noisy crying.

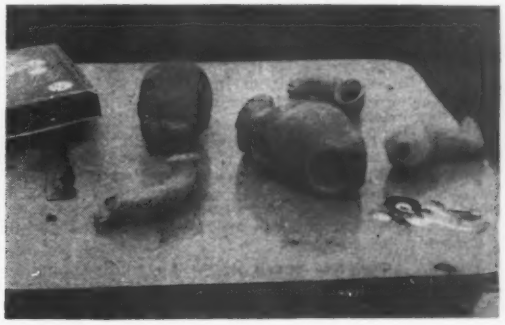

FIGURE VI.

The background to this behaviour is one of sibling rivalry. Tom was the eagerly longed-for first-born of doting parents. He became his father's constant companion. After four years twins were born. The mother's hands were busily employed in tending these usurpers. The father's aid was also enlisted, especially at feeding times, when it was his task to hold one baby while the mother fed the other. Tom felt pushed aside, as indeed he was. He lost his sense of security and privilege. The mother later insisted that he play with the twins and lend them toys. These were often broken. He found their "babyish" games very unsatisfactory. His symptoms increased until they could no longer be ignored.

Tom's behaviour difficulties are similar to others reported above—but there is no enuresis. He had been toilet trained from about twelve months onwards and had been allowed to achieve control of his bladder without being subjected to the frustration of coercion and disapproval. Figure V depicts Tom's resentment of the twins and his desire to possess his father alone. A wild elephant is killing the twins while he rides with his father in safety and watches the scene.

CASE VIII.—Dick was the son of an affectionate and painstaking mother. His training in toilet matters was achieved pleasantly and without coercion. Dick was ten years old when he reached the guidance clinic on account of backwardness at school. That his intelligence was of a very high order suggested factors other than intelligence to account for a pronounced scholastic lag.

Investigation revealed that Dick had been subjected to the intense frustration of being virtually held a prisoner among uncongenial playmates. His parents lived in a housing camp, and their neighbours, sensing their social inferiority to the newcomers, retaliated by "picking on" the family and on Dick in particular. He was "bashed" and "waited for" on his way to and from school; his week-ends were made a misery by the need of avoiding his persecutors. He was nervous, anxious and irritable, with a tendency to cry easily. We found the boy to be living apart from reality in a disquieting form of introverted thinking.

The unusual environmental factors, as cited above, were confirmed by our social worker in domiciliary visits.

Undoubtedly the frustrations in this case parallel those of the previous series; yet the child had never developed enuresis. The initial "dry bed" had been achieved in kindly fashion and without coercion.

CASE IX.—Elsie, a girl aged ten years, was brought to us because of aggressiveness, fearfulness, crying easily and tics. The child had been born after fourteen years of childless marriage. Her parents built high hopes concerning her future. Unfortunately, the child developed encephalitis with consequent retardation. Failure in school grades greatly troubled the parents. They sent her to boarding-school. At every step she was urged to a rate of progress which it was impossible for her to attain. Her sense of security was destroyed. She presented a classical case of frustration. It is noteworthy that although she developed numerous symptoms they did not include enuresis. Close inquiry revealed that the early routine of toilet training was established easily and without frustration.

CASE X.—Bobby is the five-year-old son of a woman whose husband is alleged to be suffering from war neurosis. The boy's intelligence quotient is 121. The domestic background is one of constant friction, with mood swings on the father's part and an overbearing, bullying attitude towards his wife and children. His approval is an unpredictable quality, as is also his rage.

Bobby's apathy towards school work, and his general nervousness and fear of new experiences, are the outcome of his feeling of insecurity in relation to his father. The pattern of intense frustration in the life of the child has produced symptoms which are serious, but they do not include enuresis.

It seems likely that a similar set-up in a child with a previous early and rigid toilet training would have resulted in enuresis. Bobby's mother welcomed each of her five pregnancies. Each baby was patiently and lovingly toilet trained without urging. It is interesting to note that she relates that her other children are "nervous" and easily upset, but none are bed-wetters.

CASE XI.—Anna, five years old, is the youngest child of a family of three. She has two brothers. Her mother was concerned at the child's attitude of hostility and her general intractability. She was negative, presented a feeding problem and was a cry-baby. At no time in the day did the child appear happy or content. She whined continually.

The history elicited the following facts. Anna's father has been in hospital with tuberculosis for the past year. The older brother is clever and the mother voices her pride in him often in Anna's presence. The girl is much slower, has a less attractive personality and feels eclipsed by a brother she can never hope to equal.

There is a record of toilet training which was effected easily and without stress. The initial "dry bed" was gained at about twenty months and it persisted. Ensuing frustration was powerless to disrupt the habit.

Sibling rivalry, one of the most frequently encountered disturbers of childhood security, had been reinforced in its effects by deprivation of one parent. However, this intense frustration did not result in enuresis.

Figure VI shows the kind of treatment which Anna would mete out to the brother if she could. He is dismembered and one leg has been removed. It is hidden under a box.

Conclusions.

The foregoing cases illustrate the intense frustrating process which can occur in childhood without the appearance of the enuresis dyad. There is the initial "dry bed" without the appearance of the subsequent "wet bed". The frustrating process which brought the children for treatment is of similar quality, intensity and duration. The additional factor which creates the enuresis dyad is early rigid toilet training.

Our results in treatment show that the enuresis dyad is a reversible mechanism. Patients react to treatment which is planned on total lines so as to remove the frustrating situation and restore the sense of security.

In the field of prophylaxis the way is clear. We must cease to coerce babies into "dry beds" and allow them to follow the easy, natural way which Nature intended for our children.

In conclusion, we wish to point out that the extent of the problem of enuresis has undoubtedly been underestimated in the past. Parents are ashamed to mention their child's disgrace, and many of our cases have been discovered only as a result of direct questioning.

It has been a tragic anomaly that the search for perfection by many parents has produced the bitter fruit of the enuresis dyad.

Summary.

1. The concept of the "enuresis dyad" is elaborated as one in which rigid toilet training produced an initial "dry bed". The habit breaks down under frustration to give the subsequent "wet bed".

2. An analysis is presented of 73 cases from the Child Guidance Clinic of the Brisbane Children's Hospital; 36 of the subjects are enuretic. Of these, 34 were rigidly trained in toilet matters.

3. Favourable results in treatment by the removal of frustration are recorded.

4. Examples of comparable frustrations without the "enuresis dyad" are reported. These show easy, kindly and casual toilet training.

5. The need for the abolition of strict toilet training is stressed as a prophylactic measure.

6. It is maintained that the number of enuretics in the community has hitherto been underestimated.

Acknowledgements.

Our thanks are due to Dr. Aubrey Pye, Medical Superintendent of the Brisbane General Hospital, and to Dr. D. C. Fison, Medical Superintendent of the Brisbane Children's Hospital, for permission to use the clinical material.

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"OCCUPATIONAL" FEVERS, QUEENSLAND, 1950–1951.

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THE incidence of various leptospiral and rickettsial infections in Queensland during the twelve months ending June 30, 1951, has been so great as to be worthy of record. Since most of the patients affected could attribute their infection to their work and claim workers' compensation, the group has been loosely termed "occupational". The figures presented are of cases in which the diagnoses were finalized between July 1, 1950, and June 30, 1951. This is slightly different from a record of onset during the same period. However, from accompanying figures the time of onset can be obtained by those interested. These figures show the months of onset and the number of cases of the different types of "fever" in each locality.

Many patients suspected of suffering from leptospirosis or rickettsiosis on further investigation gave negative laboratory findings. By the recording of all cases when first suspected, there is subsequently on hand a list both of patients proved to be suffering from specific diseases such as leptospiroses and rickettsioses, and of those "fever" patients whose laboratory findings were not diagnostic of any particular disease. For want of a better term, cases in the latter group have been called "pyrexias of unknown origin".

The appreciable difference in time that elapsed in some cases between date of onset and the date on which a final diagnosis was made to give an opinion for compensation purposes by one of us (D.G.) is due in the main to the distance—nearly a thousand miles—from Brisbane to the endemic areas. Most of the blood samples were sent to this department's laboratory. In quite a number of cases more than one sample was required. Occupational and sometimes clinical details had to be collected by the Weil's disease health inspectors and sent to Brisbane. Sometimes the patients, after their discharge from hospital, had moved to other districts, and difficulty was experienced in making contact with them. The existence of the patients in the first place was obtained from "Notifications of Disease", from blood samples sent in, and from field reports. This system of collecting records of cases is not perfect, but it accounts for probably 90% of the incidence.

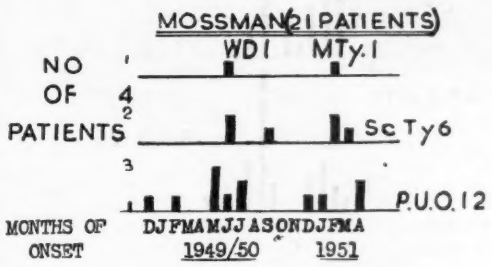

FIGURE I.

To sum up, the total number of "fever" patients investigated by the Division of Industrial Medicine in tropical Queensland was 311, made up as follows: Weil's disease, 99; scrub typhus, 49; pyrexias of unknown origin, 147; murine typhus, eight; ? tick typhus, ? murine typhus, two; leptospiroses—Pomona type, five; "Q" fever, one.

At the end of the period (June 30, 1951) there were, as well as the above, 29 additional "fever" cases which were still being investigated, but in which the diagnoses had not at that time been finalized. They represent an administrative "carry-over" to the next year, but their onset occurred in the period under discussion.

INCIDENCE IN TROPICAL QUEENSLAND.

Weil's Disease.

For the purpose of this article Weil's disease is defined as any form of leptospirosis other than that caused by the Pomona type leptospira. There were 99 cases and one death.

There were 99 patients considered to be suffering from Weil's disease. Four of these infections were diagnosed on clinical grounds only, serum from four patients gave "trace" agglutinations only, and serum from the remaining 91 gave agglutination titres diagnostic of Weil's disease. Owing to rush of work the type of leptospira was not determined in all cases. In some 29 cases so investigated *Leptospira australis A* or *Leptospira australis B* predominated; *Leptospira icterohæmorrhagiæ* accounted for about a quarter of the infections.

Of the total number of patients 92 were cane-workers, while seven had no connexion with the sugar-cane industry. The occupations of the latter were as follows: (i) Tully: two timber cutters, one slaughterman; (ii) Babinda: one timber cutter, one shop delivery man, one housewife; (iii) Gordonvale: one railway maintenance worker and pig shooter.

The canefield leptospiral infections in Queensland occur along the wet coast line from Ingham to Mossman. The annual average rainfalls for representative localities are as follows: Rockhampton 38·95 inches, Mackay 66·91 inches, Ayr 41·54 inches, Townsville 45·72 inches, Ingham 78·58 inches, Tully 178·06 inches, Innisfail 143·49 inches, Babinda 162·55 inches, Cairns 88·65 inches, South Mossman 86·57 inches, Cooktown 69·84 inches.

These show the particularly heavy rainfalls experienced at Tully, Innisfail and Babinda. Most of this rain falls in January, February, March and April, while July, August, September and October are usually "dry" months. May, June, November and December occupy an intermediate position.

Rats abound in other coastal cane-growing areas where the rainfall is less, but in these areas of lesser rainfall no Weil's disease has been found. In the northern areas of this State the cane harvest or "crushing" usually begins some time in May and finishes in December. From the point of view of Weil's disease prevention the first few months can be difficult, for in May and June heavy rain can still occur and quickly inundate land already well soaked by the "wet season" in the early months of the year. In the endemic areas excessive rain during the harvest adds to the risk of infection, particularly among cane cutters on low-lying land where slime and mud take time to dry. Usually, however, the incidence remains

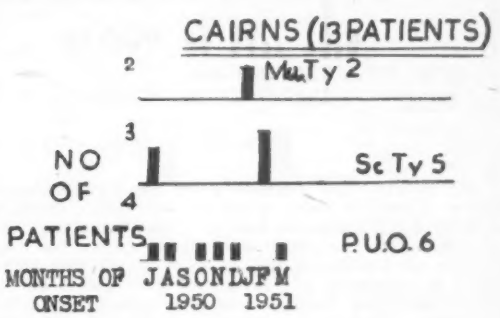

FIGURE II.

fairly low. In years of average rainfall probably not more than a dozen cases a year occur. However, the 1950 cane-harvesting season in the areas in question was preceded by unusually heavy rain in April. (For instance, in April, 1950, Innisfail's rainfall was 45·85 inches, whereas its average rainfall for April is 19·95 inches.) This was followed by an explosive outbreak of Weil's disease after rain in June (5·33 inches). After this, stricter control measures were instituted and further heavy rain towards the end of the season (23·85 inches at Innisfail in November, 1950, and 41·34 inches at Innisfail in December, 1950) did not produce a correspondingly heavy incidence of cases.

There has been the power for many years to force the burning of sugar-cane growing on low-lying land infested with rats, immediately before cutting and harvesting. In the face of this outbreak a further restrictive clause was introduced to the effect that such burns had to be efficient —that is, a mere attempt to burn cane on dangerous land in wet weather ceased to comply with the regulations. The burn now had to be a successful one. In effect, then, harvesting operations now tend to be suspended in wet weather on land where it is thought an effective burn would be difficult to obtain, for if a poor burn is obtained the farmer may not be allowed to cut and harvest the cane and it will spoil.

However, it does not necessarily follow that the lower incidence of cases towards the end of the season was due to these measures of prevention, for there are puzzling aspects of outbreaks of Weil's disease in these canefields which suggest that the number of rats and slimy, wet conditions are not the only factors of importance. Tully, Innisfail and Babinda, for instance, for practical purposes have much the same rainfall—a very heavy one—and there are low-lying, poorly drained canefields in all three districts. Yet until this outbreak Babinda had escaped any great incidence, and the fact that canecutters in the Tully

area were hardly affected is difficult to explain. Obviously among other things greater study of the amount of infection among rat populations is needed. If the amount of infection among rats is a major factor in causing a severe outbreak, then rats must confine themselves rather rigidly to distinct districts, for distances between these cane areas

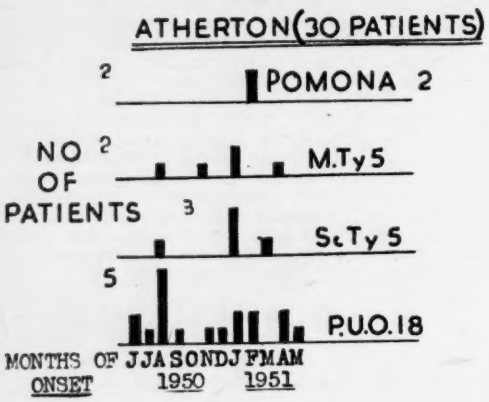

Figure III.

are not great. The distance from South Johnstone to Tully is a case in point, although admittedly there is a well-defined hill formation between these two areas. However, we can be certain of one or two facts. No matter what the rat population and its state of infection may be, rainfall is necessary, and no matter how wet it is, badly drained, low-lying farms are also necessary before many cases occur. This explains the freedom of the Mourilyan

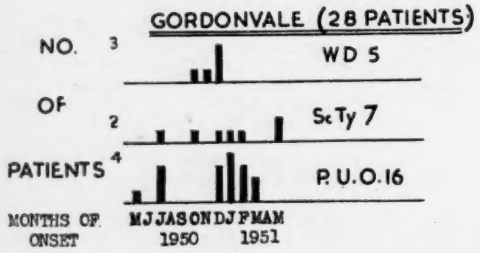

Figure IV.

area in the Innisfail district from the disease, and in reverse gives an explanation of the frequency of cases in the neighbouring Goondi area, where there are low-lying river flats.

Figures V, VI and VII illustrate the foregoing statements.

Scrub Typhus.

There were 49 cases of scrub typhus—a marked rise in the incidence experienced in previous years. In the interpretation of the scrub typhus results, however, a word of warning is necessary. Many of these patients gave agglutination titres less than one in 160 to Proteus OXK. For a variety of reasons these less significant titres were accepted as diagnostic. These reasons were usually some of the following: a fever running a clinical course suggestive of scrub typhus; a history of a period spent in scrub areas known to be highly dangerous; a definite story of bites and/or eschar and/or rash; a rapid response to "Chloromycetin". It is readily granted that other workers may have placed some of these cases in the "undiagnosed" group.

The occupational details of these patients were as follows: working wholly or partially in the scrub, 22; entering the scrub for pleasure—shooting, gathering stag-horns, swimming, walking, *et cetera*, 20; working in sugar-cane, seven.

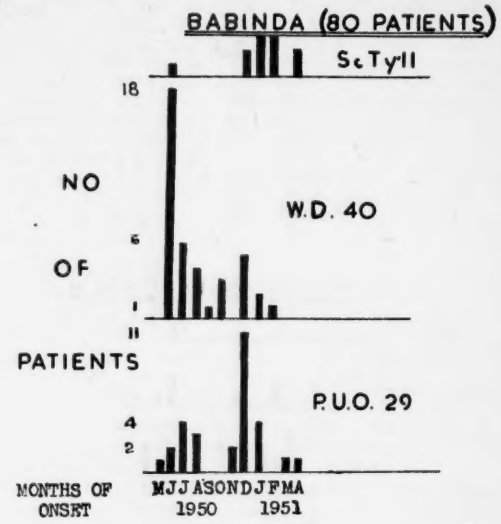

Figure V.

(Records of the movements of five of the seven cane workers were not sufficiently detailed to exclude visits to neighbouring scrub for purposes of pleasure.)

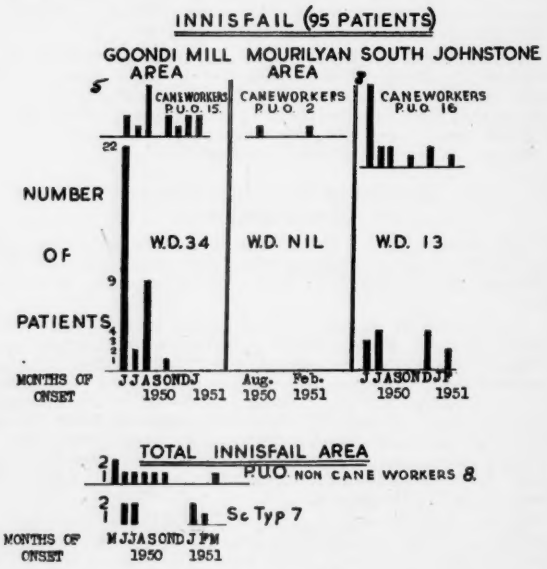

Figure VI.

Other Fevers Diagnosed.

Murine Typhus (or Tick Typhus).

The distribution of murine or tick typhus was as follows: Mossman one case, Cairns two cases, Atherton five cases, Thursday Island one case, Townsville one case. (Of these ten patients, the occupational history of eight of them indicated that their infection was that of murine typhus.

With regard to the other two, occupational and clinical details have not been sufficiently clear-cut to help very much. However, in the absence of definite pointers to tick typhus—which is rare—it is reasonable to suppose that these two also probably suffered from murine typhus.)

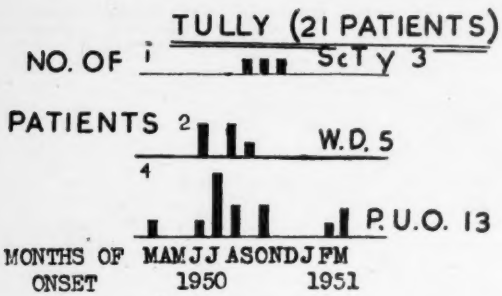

FIGURE VII.

Leptospirosis (Pomona Type).

The distribution of leptospirosis (Pomona type) was as follows. Three persons were affected at Mackay; two were dairy farmers and one was a cane farmer who kept a few cows. At Atherton two persons were affected; one was a dairy farmer and the other was a cream carter.

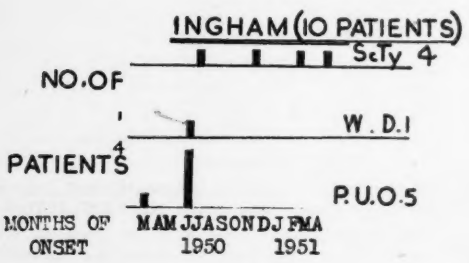

FIGURE VIII.

Thursday Island: one case of murine typhus, January, 1951. Townsville: one case of murine typhus, May, 1951. Proserpine: two cases of pyrexia of unknown origin, one each in November and December, 1950. Mackay: five cases of pyrexia of unknown origin, one in November, 1950, three in January, 1951, and one in February, 1951; one case of scrub typhus, in July, 1950; three cases of leptospirosis (Pomona type), two in January and one in March, 1951; one of "Q" fever, in April, 1951; a total of ten cases at Mackay.

"Q" Fever.

There was one case of "Q" fever at Mackay, the patient being a dairy farmer.

Pyrexias of Unknown Origin.

The number of patients whose feverish illness was not diagnosed in spite of laboratory investigation totalled 147. In the great majority of cases they were sufficiently ill to be admitted to hospital, their symptoms were not those of any common malady such as influenza, and experienced clinicians had considered the sending of blood samples many hundreds of miles for diagnosis well warranted. These facts are inserted to give a background to these patients and to show that they cannot be summarily dismissed as suffering from "colds" *et cetera*. This group of undiagnosed fevers is numerically the greatest. Any attempt at explanation would be a guess only. Their presence is at once a puzzle and a challenge to future research.

It has been pointed out to the writers that there may perhaps be some correlation between the locality incidence of Weil's disease and the locality incidence of pyrexia of unknown origin (contrast the comparative absence of either in the Mourilyan cane area). While this line of thought is tempting, the writers feel that at the moment there are so many unknown factors that it would be better to keep an open mind on the subject.

Figure IX is a graph which shows the incidence in tropical Queensland of the three principal fevers, plotted according to month of onset. In it there would appear to be some relation between the incidence of Weil's disease and the incidence of pyrexia of unknown origin. However, study of the preceding figures shows that this correlation breaks down in many of the individual localities.

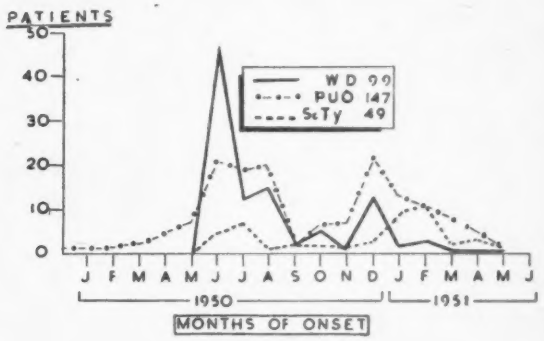

FIGURE IX.

INCIDENCE IN SUB-TROPICAL QUEENSLAND.

The winter and spring of 1950 in southern Queensland had an unseasonable and exceptionally heavy rainfall. This was followed by the usual "wet" season in the early months of 1951. As a result of this, farms—particularly in certain coastal areas where the rainfall even in dry years is usually reasonably good—became veritable quagmires. These seasonal factors may have had some bearing on the high incidence of leptospiral fevers, in particular being a factor in causing an almost explosive outbreak of leptospirosis, Pomona type, among the dairy-farming communities between Nambour and Gympie, an area in which the average annual rainfall is higher than that of most parts of southern Queensland.

Leptospirosis, Pomona Type.

Table I records the incidence of Pomona type leptospirosis. The patients in the Nambour-Gympie area were in nearly all cases living on dairy and/or pig farms. Officers of the State Department of Agriculture and Stock had investigated sickness among the cattle on many of the farms where Pomona type leptospirosis had subsequently occurred in human patients. *Leptospira Pomona* had been found in blood taken from many of these bovines when it was examined at the Animal Health Station, Yeerongpilly. With the aid of the District Inspector of Stock, a follow-up was carried out on most of the patients in the area between Gympie and Nambour. The object was to discover if any residual renal lesions had resulted. None was found. Recovery seemed to be complete. However, one of us (D.G.) carrying out this survey was struck by the fact that though the ultimate result was benign, the illness in those past adolescence could be very severe. Severe cases, however, were probably the exceptions rather than the rule. Several patients who had had malaria remarked that it was much worse than that disease. On two occasions the information was spontaneously volunteered by apparently matter-of-fact bushmen that they had thought they were going to die and almost welcomed death as a release. In adults, particularly in the odd older person, convalescence was at times prolonged and depressing.

During investigation of the farms, it was almost commonplace to hear a farmer state that after a cow had become

sick and her milk had "dried off", he had sold her as a "tinner" to a dealer, to be subsequently slaughtered for meat of somewhat inferior quality.

The heaviest incidence among the farming community was in January and February. The Brisbane cases, chiefly among abattoir workers, occurred in March, April and May. It may be pertinent to wonder if there is any connexion between these events. The odd case occurring in the drier districts (Nanango, Chinchilla *et cetera*) has always been a feature of the incidence in this State.

It is a matter of interest that on one farm the husband was very sick with leptospiral (Pomona type) infection and some months later his wife had brucellosis.

TABLE I.
Pomona Type Leptospirosis.

Month.	Brisbane Area, Including Ipswich: Number of Cases.	Nambour to Gympie: Number of Cases.	Elsewhere: Number of Cases.
1950:			
July	1	—	
October	—	1	
December	—	2	Chinchilla .. 1
			Gayndah 1
1951:			
January	1	11	Chinchilla .. 1
February	1	21	Nanango 1
March	4	—	Chinchilla .. 1
April	4	2	Gladstone .. 1
May	3	2	Gladstone .. 1
June	—	3	Chinchilla .. 1
Total	14	42	8

Infections with Leptospira Formerly Termed Mitis—Designation Now Under Review.

Table II shows that in districts with heavy infections from *Leptospira Pomona* there were also a few cases of infection with the strain formerly called *mitis*.

TABLE II.
Infection with Leptospira (Formerly Termed Mitis).

1951.	Nambour-Gympie: Number of Cases.	Gladstone: Number of Cases.
January	1	—
March	1	—
May	—	1

"Q" Fever.

The incidence of "Q" fever was higher than that noted for some years past. Nearly all the patients were connected with the slaughter of cattle. Table III shows the time and place distribution.

Brucellosis.

Seven patients were proved to have brucellosis—the type was not determined. The occupations in some cases do not clearly indicate whence the infections came.

Comment.

To complete the picture, it must be pointed out that in subtropical Queensland also there are a number of "fever" cases, the investigation of which produces negative results. The clinical problem of the pyrexia of unknown origin therefore is not confined to North Queensland, but is particularly noticeable there. The writers have stressed the northern aspect simply because it was their duty to investigate superficially the problem there, and not because they regard the incidence elsewhere as of no importance. Since this article was written the Queensland Institute of Medical Research has established a Field Station at Innisfail to investigate the problem of fevers in northern Queensland.

TABLE III.
"Q" Fever.

Month.	Brisbane Area: Number of Cases.	Nambour-Gympie: Number of Cases.	Gladstone: Number of Cases.
1950:			
August	1	—	—
September	1	—	—
December	4	1	—
1951:			
March	1	—	—
April	6	—	—
May	1	—	1
June	4	—	—
Total	18	1	1

TABLE IV.
Brucellosis.

Month.	Type of Worker: Number of Cases.	
1950:		
December	Abattoir worker	1
1951:		
January	Abattoir worker	1
	Farm housewife (? raw milk)	1
March	Printer (drank raw milk)	1
April	Grazier (drank raw milk)	1
May	Abattoir worker	1
June	Dairy-farm housewife	1

Summary.

1. A high incidence of various leptospiral and rickettsial fevers occurred in Queensland in the years 1950-1951.

2. In North Queensland there were 99 cases of Weil's disease—associated mainly with the sugar-cane industry—and 49 cases of scrub typhus. As well as this there were 147 patients presenting with severe fever for which a satisfactory explanation was never found in spite of investigation. Occupational histories were obtained from these patients. There were also 10 cases of tick or murine typhus, five cases of leptospirosis (Pomona type), and one case of "Q" fever—in all, 311 patients.

3. In southern Queensland, mainly among dairy and pig farmers and men connected with the slaughter of livestock, there were 64 patients infected with *Leptospira Pomona*, three affected with the strain formerly called *Leptospira mitis*, and 20 patients affected with *Coxiella burneti*. As well as this, seven people suffered from brucellosis. In southern Queensland the writers did not keep a record of cases of murine typhus or of patients whose investigations gave negative results.

4. It has been suggested that exceptionally heavy rainfalls may have influenced the unusually high incidence of all these fevers.

Acknowledgements.

The authors wish gratefully to acknowledge the interest and cooperation of the many medical practitioners throughout the State who supplied the clinical records, and who in many cases cheerfully took numerous blood samples. Thanks are due to the staffs of the Commonwealth Laboratories at Cairns and Townsville, and to the staff of the Laboratory of Microbiology and Pathology, Department of Health and Home Affairs, Brisbane, for carrying out laboratory investigations. Thanks are also due to Dr. E. H. Derrick and Dr. D. W. Johnson for much sage advice, and to the Director-General of Health and Medical Services for permission to publish this article.

THE MOVABLE EYE IMPLANT: A DESCRIPTION OF THE IMPLANT TECHNIQUE USING THE CUTLER-HAMBLIN PROSTHESIS.

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Introduction.

THAT the cosmetic appearances after simple enucleation of an eye, followed later by the insertion of a glass or plastic eye, often leave very much to be desired is well known. Frequently the deformity is easily noticed from the sunken appearance of the socket, the fixity and stare of the artificial eye and the fact that, particularly in the course of ordinary conversation, it is only one eye which is seen to undergo the small-amplitude but rapid flicks of movement normally carried out.

Many people become socially unacceptable or retire from social contacts as a result of this operation.

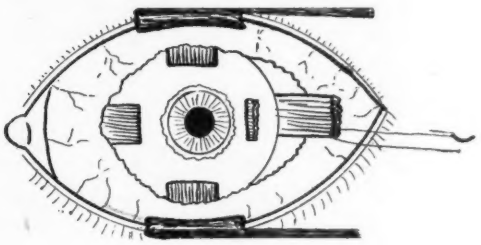

FIGURE I.
The lateral rectus tendon has been cut after insertion of the suture.

Historical.

In 1579, Paré mentioned an eye of gold being worn by a man who had lost an eye. The modern glass eye was first made in 1878, while plastic eyes have appeared within the last ten years. In 1941, a semi-buried implant was first extensively tried out, and the introduction by Cutler of his implant with tantalum mesh for fixation of the muscles and a hole for a peg, which is attached to the plastic eye, marked the first really successful advance.

General Principles.

At the time of enucleating the eye (or later in suitable cases) the implant is inserted into the orbit within the muscle cone, and the four recti muscles are attached to the mesh on its antero-lateral aspect (Figure V).

The conjunctiva is sewn around the margins of the flat anterior surface of the implant, which remains exposed. As the recti still retain full movement after the eye itself has been excised, the implant will move in a manner similar to the other eye.

When this is healed into position, an artificial eye is placed in position. This artificial eye has a peg attached to its posterior surface which fits into the hole on the anterior surface of the implant; thus the movements of the implant are transmitted to the artificial eye.

Technique.

The operative technique has been modified several times in the author's series, this being the final form as employed at present.

A general anæsthetic is administered and the operative field prepared.

Stage I: Enucleation.

The conjunctiva is incised at the limbus and Tenon's capsule is well separated from the sclera.

To one side of each of the tendons of the recti muscles adjacent to their insertions a tantalum wire suture is placed, and the muscle is freed from Tenon's capsule as

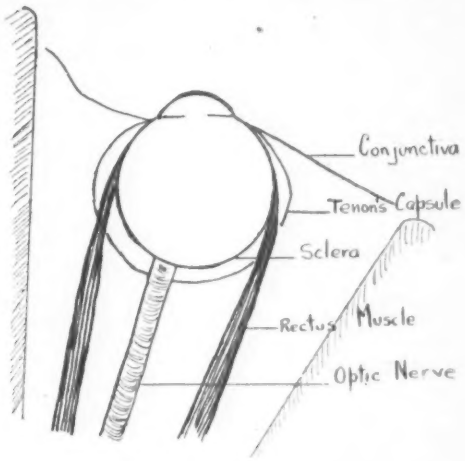

FIGURE II.
Normally placed conjunctiva, Tenon's capsule, recti muscles and optic nerve.

far back as the muscle opening in Tenon's capsule. The tendons are now cut at their insertion into the sclera and the wires are clamped at the appropriate quadrants to avoid crossing or rotation of the muscles (Figure I). Tantalum wire is used, as catgut soon softens, leaving the ends of the tendons loose, the muscles being held only

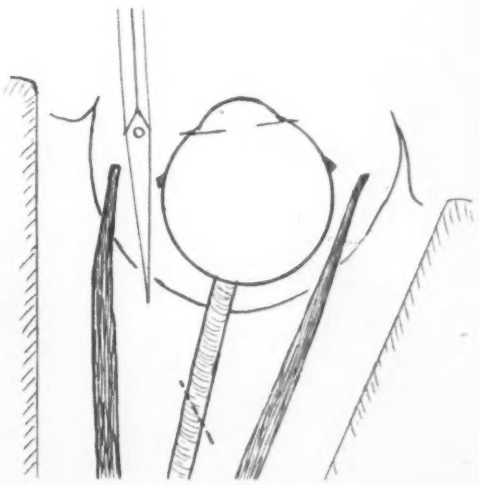

FIGURE III.
Tenon's capsule is incised far from the optic nerve.

by the second line of sutures; this has been seen on two occasions when the anterior part of the implant was examined eight and eleven days respectively after insertion.

The superior oblique tendon is caught by a strabismus hook passed laterally across the top of the globe, the end pointing laterally. It is cut near its insertion and the

tendon followed medially to the trochlear region, where it is again cut, as much having been pulled through as possible. If this is not done, the strong tendinous band may attach itself to the fibrous tissue on the implant and pull it forwards, as was seen in one case, with loss of upward movement and near extrusion (Case I).

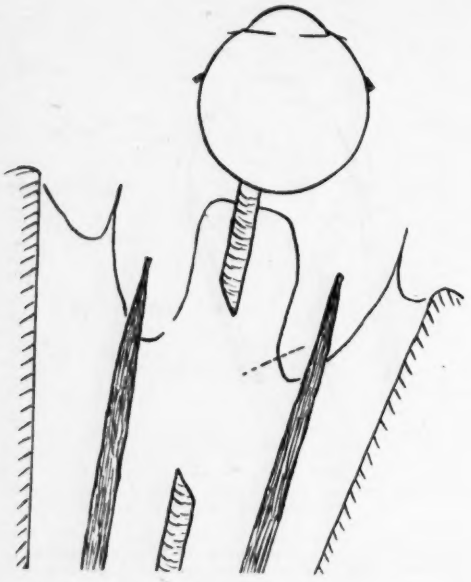

FIGURE IV.
Shows the inverted portion of Tenon's capsule to be excised.

The inferior oblique is resected by catching it near the insertion, cutting the muscle, pulling and cutting again, care being taken not to damage the inferior rectus, under which it passes whilst in its fibrous tissue sheath.

The excision is completed by removing with the stump of the optic nerve as much as possible of the posterior portion of Tenon's capsule by the following method. A representation of the relevant tissues is seen in Figure II. A hole is made in Tenon's capsule posteriorly as far from the optic nerve as possible, and through this hole the optic nerve is severed with straight scissors passed from the upper and inner quadrant, not too far past the optic nerve to avoid injury to the rectus muscle on the opposite side (Figure III). The globe now comes forward, attached by only an incomplete apron of Tenon's capsule inverted by its attachment to the base of the optic nerve which is still with the globe (Figure IV). This is cut as widely from the optic nerve as possible. This technique amounts to a resection of the posterior portion of Tenon's capsule, this allowing the implant to fall back into the orbital fat and not to be extruded by subsequent contraction of Tenon's capsule fibres.

The socket is now packed with gauze strips until hæmostasis is complete, persistently bleeding vessels being ligated. (This latter course was necessary in only one of 14 cases.) Sufficient time to obtain a bloodless field to complete the operation only is needed, as further packing and bandaging later preclude a serious secondary or reactionary hæmorrhage.

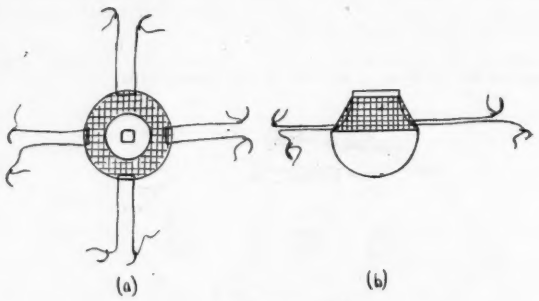

FIGURE VI.
The posterior sutures placed in the implant.

Stage II: Implantation.

The implant is shown in Figure V.

Ten-inch double-armed lengths of size 0 silk with size 6 eye needles are passed through the posterior third of the mesh on the four quadrants, the distance between the two arms of one length being about half the width of the tendon (Figures VIA and VIB).

The implant is now placed within the muscle cone.

The two needles are passed through the middle third of the tendons or muscle four to five millimetres from the

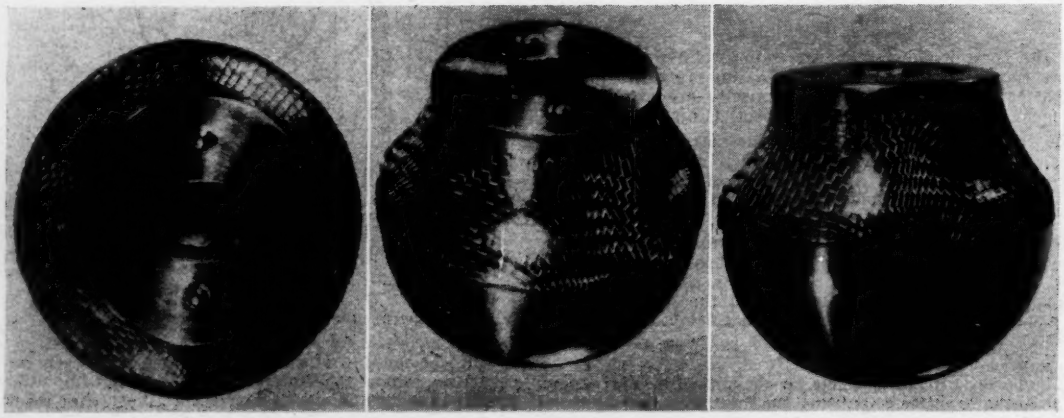

FIGURE V.
Photographs of the implant (from left to right): front view, oblique view, side view.

severed ends, and continued through the conjunctiva about 10 millimetres from its cut edge, the double ends being held in clamps (Figure VII).

The tantalum wire is passed through the mesh in its middle area at positions to correspond with the final positions of the ends of the tendons, passed through the other side of the tendon, and tied; the knot is pushed beneath the crossing wire.

The silk sutures are now tied, care being taken to pull the conjunctiva well towards the centre, and are cut; free ends measuring three-quarters to one inch are left beyond the knot to facilitate their later removal (Figure VIII).

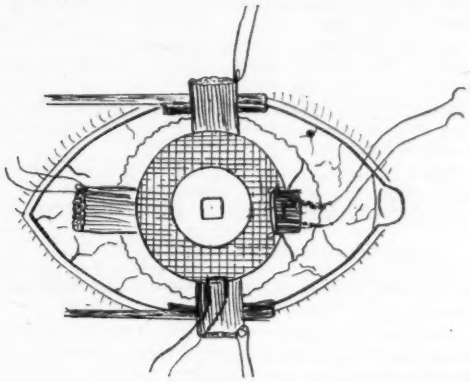

FIGURE VII.

The medial rectus has been sewn to the mesh. The posterior sutures have been passed through the medial and inferior recti muscles.

The conjunctival edge is now sewn to the anterior portion of the mesh by a continuous over-and-over stitch, the edge of the conjunctiva being inverted to lessen the possibility of granuloma formation. Size 0 silk or tantalum wire is used. The flat anterior surface of the implant containing the square peg hole is left exposed (Figure VIII). Figure IX demonstrates the relation to one another of the three sutures in each muscle.

Stage III.

The conjunctival sac is packed with *tulle gras* to ensure that there is no early shrinkage of the conjunctiva, and double eye pads are placed in position, that over the implant being bandaged firmly, the other maintained with strapping. (It is not unknown even for the prepared patient to have torn off the covering of his good eye just to make sure!) This pad can be lifted as the patient comes out from the anæsthetic and replaced without the implant dressing being disturbed. The patient is given prophylactic penicillin therapy and the double eye pads are maintained in position for seven days (the outer dressing being changed daily); the patient is allowed a peep at the world on occasions.

On the seventh day the conjunctival packing is removed, a painless procedure, and a large flat retaining shell is slipped into the conjunctival sac, one without a peg being preferred so that the implant can move freely behind it. No dressings are required and the patient gets up and moves about. If the discharge from the socket is minimal he leaves hospital on the tenth to fourteenth day.

The silk sutures are removed on the twenty-fifth to thirtieth day, any residual discharge usually ceasing when this has been done.

The artificial eye of plastic is placed in position for comparison purposes in four to five weeks from the date of implantation, and the finished eye is left *in situ*; it is removed only if there is any reason for doing so—for example, for inspection by the surgeon.

Typical Case Records.

CASE I.—A boy, aged twelve years, injured his left eye, and in February, 1950, the blind unsightly eye was excised and a Cutler-Hamblin implant inserted. The result was satisfactory until August, 1950, when the left eye became progressively prominent and upward movement diminished. In November, 1950, at operation, the superior rectus was found almost to have lost its attachment to the mesh; the superior oblique was attached to the implant, and contraction of Tenon's capsule did not allow the implant to return more than a small distance into the orbit. The superior oblique tendon was resected, as well as two segments of Tenon's capsule. The implant fell back well, and subsequent follow-up

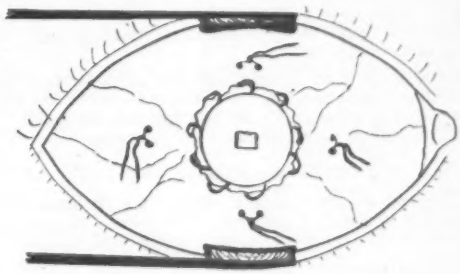

FIGURE VIII.

The appearances after all sutures have been tied.

examination after three months showed only an eye pleasing in appearance, with good movements and comfortable to its wearer.

CASE II.—A man, aged twenty-six years, suffered an expulsive hæmorrhage from his right eye following an industrial accident. Implantation was followed by an uneventful course. Three months later it was difficult to tell which eye was artificial unless the eyes were very closely inspected. Move-

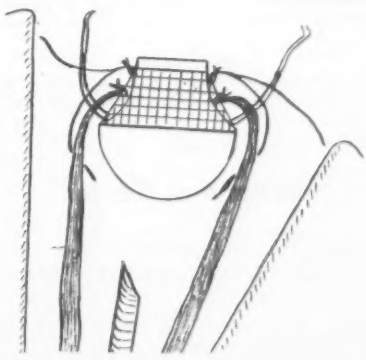

FIGURE IX.

The relative positions of the sutures are demonstrated.

ment was very good, there was no discomfort and no discharge. He had learned how to converge both eyes when looking at close objects.

CASE III.—A woman, aged thirty-six years, had her left eye excised, a choroidal malignant melanoma being present, and an implant inserted. Subsequent examination of the eye did not reveal any extraocular extension, and the cosmetic result left little to be desired in this very good-looking woman, who continued her social activities without the usual resort to dark or heavily rimmed glasses and without anyone noticing her defect.

Complications.

Complications which may be met are numerous, but not commonly seen, and may be prevented in great measure

by an adequate technique. They include persistent discharge, pain, loss of movement in one direction, squint, granuloma formation and extrusion.

Results.

The appearances so resemble a normal pair of eyes that difficulty may be encountered in telling which is the operated side.

The psychological effect on the patient is enormous. A dreaded deformity is not present and nothing unusual is noticed by his or her fellows.

Pathological Considerations.

It has been shown that epithelium grows behind the implant and that foreign body reaction in the orbital tissues is almost absent.

Certain it is that in the absence of infection and of contracting bands and sheets of fibrous tissue, the implant is very firmly adhered to by the tissues, as much to the smooth tantalum surface as to the meshwork.

Conclusions.

1. The use of the implant marks one turning point in ophthalmic surgery, a deforming procedure being replaced by one æsthetically pleasing and socially acceptable.

2. For a successful result in the highest proportion of cases, attention to technical minutiæ, each of which has a definite reason for its adoption, is important; this includes post-operative management.

Summary.

The great desirability of replacing a deforming operation with one which leaves the patient's appearance near to normal is put forward. The technique is discussed in full, the details being of considerable importance.

Acknowledgements.

Acknowledgements are made to the surgeons of the Moorfields, Westminster and Central Eye Hospital, London, for most liberal teaching and practical experience.

Reference.

Guyton, J. S. (1948), "Enucleation and Allied Procedures", *Transactions of the American Ophthalmological Society*, Volume XLVI, page 472.

Reports of Cases.

BITE FROM BROAD-HEADED SNAKE: HOPLOCEPHALUS BUNGAROIDES (BOIE).

By H. FLECKER,
Cairns, North Queensland.

THE victim's name is given in full in this contribution with his full knowledge and consent. He is an enthusiastic herpetologist, and he had a keen personal interest in the snake concerned, of which he supplied the determination; he is also responsible for supplying most of the clinical details, particularly of objective and subjective symptoms, especially for the initial three hours prior to his admission to hospital. Apparently no clinical notes of the bite from this species have yet been published in any medical journal.

He had been bitten by a sand snake, *Demansia psammophis*, a native of North Queensland, in June, 1951; for this he had been treated as an out-patient at the Cairns Base Hospital, and had received tiger-snake antivenene. The broad-headed snake, *Hoplocephalus bungaroides* (Boie), is not found in North Queensland, but a specimen was brought by him from Sydney for study. This he kept in a small herpetarium.

Clinical Record.

William Hosmer, junior, aged twenty-six years, apparently in good health, was bitten at 4.35 p.m. on November 26, 1951, on the palmar aspect of the middle finger of his left hand by this same broad-headed snake, which was two feet five inches in length, whilst he was engaged in replacing drinking water in its cage. The bite was followed by a stinging sensation in the bitten area. A rubber ligature was placed above the elbow and another above the distal interphalangeal joint of the finger attacked. Surplus venom was wiped away, the fang punctures were incised, and this procedure was followed by suction from a rubber suction bulb.

At 4.45 p.m. the finger was greatly swollen, and by 4.57 p.m. the swelling was evident over the knuckles and the back of the hand.

At 5.5 p.m. the tourniquet was released for thirty seconds. The patient felt normal, apart from the swelling, and went about his duties feeling well.

At 5.35 p.m. the ligature was again released for thirty seconds, the one applied to the affected finger having been discarded altogether.

At 5.45 p.m. the arm had swollen somewhat, and as the patient complained of severe headache, hot tea was given with a generous sugar content, together with three aspirin tablets.

At 6.5 p.m. the ligature was loosened for thirty seconds, but headache was still present, although not so severely as before. There was swelling in the left shoulder and the arm was stiff and sore to the touch.

At 6.35 p.m. the ligature was released for thirty seconds.

At 6.53 p.m. he felt light on his feet as though slightly intoxicated, his eyes being glassy and his face flushed. Vomiting occurred three times between 6.45 and 7 p.m., and he was compelled to lie down.

At 7 p.m., when his condition became obviously worse, I was sent for; I immediately drove him to the Cairns Base Hospital, where he was admitted.

The following notes are supplied through the courtesy of Dr. B. Geaney, the resident medical officer. Upon the patient's admission to hospital, the arm below the tourniquet was red, swollen and painful. Ten millilitres of tiger-snake antivenene were injected intravenously; seven minims of adrenaline were given about thirty minutes later; phenergan, 25 milligrammes three times a day, was also prescribed.

At 9.30 p.m. the tourniquet was removed.

At 11.15 p.m. the left arm was very swollen and painful. The pulse rate was 108 per minute and the temperature was 99·2° F.; one-sixth grain of morphine was given.

At midnight the arm was still very painful. The pulse rate varied considerably between 48 and 72 per minute, and the blood pressure was 105 millimetres of mercury, systolic, and 50 millimetres, diastolic. The patient was complaining of dizziness and of aches and pains in the joints—probably a slight serum reaction.

On November 27 his temperature was 101·5° F. and the pulse rate was about 100 per minute. The patient was feeling ill; he had pains in the arms and legs and headache. His left arm was red, swollen and painful. The administration of penicillin, 300,000 units twice daily, was commenced.

On November 28 his temperature was 102° F. and his pulse rate about 100 per minute. He was more comfortable, but his arm was still very swollen.

On November 29 his temperature was 99° F. and his pulse rate 80 per minute.

On November 30 he was discharged from hospital.

The following additional notes have been supplied by the patient.

On November 27 he was unable to open his mouth widely and had some difficulty in swallowing. He was unable to move the lower limbs at all, and the uninvolved part of the left arm could not be raised above the level of the bed. There was also some stiffness of the muscles of the neck which prevented free movement in this region.

On November 28 the disabilities of opening the mouth and of swallowing disappeared, but those of the left arm and lower limbs were less pronounced. He was able to move his toes, which previously he could not do.

On November 29 the above-mentioned disabilities had mostly disappeared. He was allowed out of bed for fifteen minutes, during which time he experienced considerable difficulty in walking.

The patient had previously been bitten in Sydney by the same specimen of broad-headed snake. The reptile had just been "milked" of its venom. The effects, though painful, caused symptoms of headache, drowsiness and nausea, but no vomiting. The effects lasted for twenty-four hours, and the hand was swollen for three days. The symptoms were evidently curbed by the small amount of venom injected (due to the "milking"), and owing to the immediate first-aid treatment no admission to hospital was thought necessary.

Whether the initial dose of venom injected by the snake in Sydney, or the tiger snake antivenene administered in June, 1951, conferred any immunity is difficult to determine from the above account. It certainly was not complete in any case.

COMMENTS ON ACUTE APPENDICITIS OF CHILDHOOD.

By FREDA GIBSON, O.B.E., M.B., B.S. (Adelaide),
Ceduna, South Australia.

AFTER reading Dr. E. Stuckey's article on appendicitis in childhood in THE MEDICAL JOURNAL OF AUSTRALIA, December 15, 1951, and looking back on my nineteen years in private practice, I was struck by the similarity of my own findings and treatment of the various aspects of this rather difficult condition.

I note, however, that my district has a greater awareness of the urgency of consulting the doctor about any complaint of abdominal pain in children than appears to be the case in Sydney. In spite of the fact that my practice covers a very large outback area and the people sometimes have no telephone communications, the majority make contact with me within the first twenty-four hours of the onset of abdominal pain, and often within twelve hours. This is partly because I have been the family doctor here for eighteen years and have therefore had the opportunity to educate the people on the importance of early medical advice for any abdominal abnormality.

I am able to quote almost identical figures for the relative frequency of the various positions in which the appendix is found on operation ,and heartily endorse Dr. Stuckey's classification of appendix types.

With regard to symptoms, I have found that anorexia is nearly always present at the commencement of an attack. One may have to search for this symptom, as often it is not sufficient to ask whether the child ate its last meal. On further inquiry the mother will admit that "she just ate her 'Granose' but refused her toast and egg". The absence of this symptom is of greater significance than its presence in helping to exclude a diagnosis of appendicitis.

Dr. Stuckey's observation that urinary symptoms occasionally confuse the history recalls to me the following case.

A female child, three years of age, had a history of having "gone quiet" for the previous ten months, with loss of appetite. She had suffered one attack of abdominal pain with vomiting four months before, when she also had acute coryza.

When I first examined her she had these positive signs: a temperature of 101° F., a pulse rate of 140 per minute, a respiratory rate of 28 per minute, urine containing a slight cloud of albumin. Her abdomen was distended and tympanitic above the umbilicus and down each side, but dull below; this dulness was obviously due to a full bladder. The presence of muscle rigidity was difficult to assess. On rectal examination with the tip of my finger I could just feel something hard through the anterior wall of the rectum, which I thought might be a pelvic mass or scybala in the sigmoid colon. The white blood cell count was 4500 per cubic millimetre.

An enema was given with a constipated good result and the child micturated, when I found that the abdominal distension and tenderness and the pelvic mass had all disappeared. One hour later another motion was passed, the temperature fell to normal and the patient felt quite well.

Six weeks later the symptoms and signs returned and on rectal examination the mass could be felt as before, only larger, being now the size of a hen's egg. X-ray examination with a barium meal revealed no abnormality. The white blood cell count was 8750 cells per cubic millimetre. On abdominal examination the bladder was again full and bowel distension was present, but on micturition the abdomen became normal again. The temperature remained slightly raised (99·5° F.). The pelvic mass also remained.

I referred her to Dr. W. Jolly in Adelaide, and he made a diagnosis of appendicitis. On laparotomy the appendix was found to be hanging over the pelvic brim and the pathologist reported a pathological appendix. No other abnormality was found and convalescence was uneventful.

In this case the possibility of appendicitis was constantly in my mind, but I was unable to confirm the diagnosis because of the rapid disappearance of the symptoms and signs. Dr. Jolly, however, obtained tenderness over McBurney's point.

Another interesting case I had a few months ago was a case of true acute mesenteric lymphadenitis, stated by Mr. Stuckey to be an undoubted clinical entity.

The patient was a female child, six years old, and was not examined by me until the attack of abdominal pain was of sixty hours' duration, as she had come from another district. Her mother reported that she had been vaguely "off colour" for about two months. Her present history was that sixty hours before she had complained of central abdominal pain, passing up to the right hypochondrium; she had anorexia and had vomited once, ten hours after the onset of the pain. Her bowels were regular in action. She had complained of a similar attack six weeks earlier, when a diagnosis of tonsillitis was made.

On examination of the patient the temperature was 100° F., the pulse rate was 90 per minute, the urine was normal. The throat and tonsils were hypertrophied but not inflamed; the posterior pharyngeal lymphoid tissue was hypertrophied and inflamed. There was tenderness on palpation of the abdomen over the umbilical area, and this could be followed up to the right hypochondrium. There was doubtful rigidity in the right flank. There was no tenderness or mass palpable on rectal examination. The white blood cell count was 10,050 cells per cubic millimetre.

I started her off on a course of "Procillin" and sulphamerazine. Here temperature fell to normal on the first evening and the pain was easier.

Thirty-six hours after admission to hospital her temperature rose to 100° F. again and she complained of increased abdominal pain and tenderness. She was also tender over McBurney's point, so I decided to explore her abdominal cavity.

I chose a gridiron incision, as the diagnosis of appendicitis seemed assured.

The appendix was located in the anterior position, bound down to a very much thickened mesentery. The appendix itself was a little tense but not inflamed, and there was no kinking or obstruction. All the mesenteric glands were considerably and evenly enlarged, with a uniform pink appearance. The appendix was removed together with an enlarged mesenteric lymph gland.

The pathologist reported as follows:

> Appendix—mucosa intact, the lymphoid tissue is hyperplastic; there is no evidence of acute appendicitis. Gland—it is enlarged by reactionary hyperplasia; the follicles are prominent and some show toxic necrosis of their germinal centres.

While awaiting the pathologist's report I had carried out a Mantoux test, the result of which was negative, and a complete blood examination, the result of which was normal except for a moderate, relative increase in lymphocytes in the differential count.

The child's convalescence was uneventful, the temperature falling to normal after four days, and there has been no return of her symptoms and signs.

Lastly, Dr. Stuckey stated that he had not seen a proven case of pylephlebitis. I believe that I came across such a case a few months ago. The patient was an adult woman, aged thirty-eight years. She came to me with right-sided abdominal pain, of which she had had three attacks in two weeks. On vaginal examination I found a movable tender mass the size of a tennis ball in the right fornix. I could also push this around on abdominal palpation, as it formed a swelling in the right iliac fossa. There was slight rigidity and tenderness here also.

I made a diagnosis of ovarian cyst and on laparotomy I found the mass to be the ileo-caecal junction and the appendix completely enwrapped by omentum, with an absence of any adhesions between the mass and surrounding tissues, so that the tumour could be moved about the abdominal cavity with complete freedom. As the whole inflammatory mass was sealed off and could not be unsealed without harm to the patient, I sprinkled 15 grammes of penicillin-sulphanilamide powder around the mass and closed the abdominal cavity. Intramuscular penicillin and streptomycin therapy and oral sulphamerazine therapy were commenced.

Her convalescence was uneventful until the tenth day, when her temperature chart showed a slight rise from 99° to 99·2° F. On the tenth and eleventh days her temperature rose to 102° F., but the patient had no other symptoms or signs. On the twelfth day her temperature shot up to 105·4° F. and the patient had a rigor. She had no other symptoms.

On examination I found nothing abnormal in the chest, either clinically or radiologically, and I found nothing abnormal in the abdomen except a palpable, slightly tender liver and, of course, the movable tumour, which on gentle palpation appeared to be a little smaller than before. I made a diagnosis of ascending pylephlebitis and the penicillin, streptomycin and sulphamerazine therapy was continued. The next day I made a white blood cell count and found the number to be 11,000 cells per cubic millimetre.

The patient continued with rigors for five days, the daily temperature reaching 105° F. On the fifth day the hæmoglobin value was 70% (Sahli) and the white blood cell count was only 5000 cells per cubic millimetre. The patient now developed a twitching of her face and limbs, mental aberration and a moderate degree of deafness. She made no complaint of a sore tongue, but when I examined her mouth I found that she had glossitis and pharyngitis. I discontinued the sulphamerazine and the streptomycin, but continued the penicillin. Her progressive daily white blood cell counts from this time were 4900, 4100, 5300, 6000, 7500 and 8100 per cubic millimetre, with neutrophile cell counts of 36%, 33%, 40%, 45%, 49% and 60% respectively. The hæmoglobin value remained a constant 70% (Sahli).

No treatment was given for the agranulocytosis except the discontinuance of the sulphamerazine and the continuance of "Procillin". The patient's temperature fell to 100° F. after five days of rigors and two days later was normal. Twelve days after the first rigor the patient looked and felt well. She was given a full diet with additional iron and permitted to leave hospital in four and a half weeks.

On her discharge from hospital I could still feel a fullness in the right iliac fossa, but no definite tumour. Deep palpation caused some tenderness.

I urged the patient to report the return of abdominal pain, and this occurred ten weeks after the laparotomy. On examination of the patient I could find no definite signs of a "flare-up", and as I felt that the job of removing the appendix was still likely to prove difficult I decided to treat her conservatively, if possible.

The pain proved to be a false alarm and she had no further trouble. I readmitted her to hospital fourteen weeks after the original laparotomy and found on reopening her that the omentum had almost completed its unwrapping process; with a little gentle stripping the appendix was exposed. The tip was gangrenous for about half an inch, the gangrene beginning from a point of rupture. I removed the appendix, sprinkled a further 15 grammes of penicillin powder around the area and closed the abdomen.

The patient's convalescence this time was uneventful.

Reviews.

ANÆSTHESIA.

THE publication of a second edition of J. A. Lee's "Synopsis of Anæsthesia" is of interest to all anæsthetists.[1] Like its forerunner, this edition is of handy size and is printed in small, clear type upon good paper. Misprints are very few, but, in the copy before us, pages 297-324 have been bound out of sequence. The illustrations still tend to show only the exterior of apparatus, which gives no clue to its internal structure and makes a text-book appear like an instrument-maker's catalogue.

The second edition is larger by 100 pages than its predecessor. The new material is timely and covers all the major advances in anæsthesia, with the exception of the hypotensives. The anatomical introduction (Chapter II) has been expanded. The parts of this chapter which deal with respiration have been revised and now include a section upon collateral respiration. Chapter III is the gainer by a new section upon the pre-operative nutritional care of the patient. The account of the physiology of the autonomic nervous system (Chapter XV) has been expanded. A useful section upon electrolyte balance has been added to Chapter XXI. Anæsthetic technique for the newer cardiac operations is discussed in Chapter XXII. The muscular relaxants (Chapter XVII *et passim*) receive more attention than in the earlier edition. The organization of an anæsthetic out-patient clinic is indicated in Chapter XXVII. This small volume is indeed encyclopædic in its range.

The arrangement of chapters is, perhaps, not always happy. It is difficult to follow cognate subjects through the text. For example, the chapter on analeptics (XIV) is interposed between discussions of thiopentone and of spinal analgesia. It might belong more logically to the consideration of shock (Chapter XXI). The last-mentioned chapter, in turn, might well have been linked to that (IX) which deals with the accidents of anæsthesia. The management of the unconscious patient (Chapter XXV) might usefully have been considered earlier in the book.

Some views, from which we dissented in the earlier edition, have been reproduced unchanged in this one. An instance is the use of carbon dioxide as a respiratory stimulant, even in patients depressed by morphine (page 32) or undergoing induction with chloroform (page 65). It is stated on page 104 that "partial rebreathing should be employed in all but short nitrous-oxide-oxygen anæsthesias", a statement from which most anæsthetists of the present day would dissent. On page 124 is found the laboratory dictum that a raised tension of inspired carbon dioxide aids the liberation of oxygen from hæmoglobin, a dictum of which the clinical application is likely to lead to disaster. There occurs on page 74 the curious statement, found also in the first edition, that expansion of the reservoir bag during carbon dioxide absorption anæsthesia is a useful sign of exhaustion of the soda lime. On page 105 appears a detailed account of secondary saturation, a technique which might now be allowed to pass into decent oblivion.

We are not in accord with many of the views expressed in the text. Insufficient emphasis is placed upon the corollary to the work of Dripps, namely, that when tidal respiration becomes depressed, the mere giving of oxygen is not an adequate remedy. It must be combined with manual "aid" to respiration, designed to reproduce a normal tidal

[1] "A Synopsis of Anæsthesia", by J. Alfred Lee, M.R.C.S., L.R.C.P., M.M.S.A., D.A., F.F.A.R.C.S.; Second Edition; 1950. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 4¼" × 7¼", pp. 368, with 66 illustrations. Price: 15s.

excursion for that patient. This point is of great importance in this era of muscular relaxants. Of the latter, it is stated on page 252 that "since the use of the curare drugs became general, the need for endotracheal intubation has decreased". We hold the exactly contrary view. The text does not mention the depolarizing action of certain drugs upon the neuromuscular end-plate, although this action is important in respect of the dosage of decamethonium and of "Prostigmin". The dosage of tubocurarine for bronchoscopy in children is given on page 269 as 0·25 to 0·5 milligramme per pound of body weight, which appears to be thrice the usual dosage.

The technique for giving ether by the "open" method (page 49) would not be approved in Australian teaching hospitals. Neither would that for the administration of nitrous oxide-oxygen for minor dental operations (page 114) gain favour in our dental schools, where emphasis is put upon maintenance of an atmospheric percentage of oxygen, even at the price of minimal volatile supplement. On page 177 appears the statement that "any drug raising the blood-pressure removes one of the great advantages of spinal analgesia—it spoils the bloodless field". We dissent from this statement; we do not care for this form of "hæmostasis by circulatory asthenia". In any event, the ability of analeptics to produce sustained rise in blood pressure in such cases is problematical. Mention is made (page 173) of the fact that certain workers have failed to find oximetric evidence of hypoxia in "high" thoracic block. The dependability of the oximeter is, however, not yet generally accepted, and the author is probably wise to advocate oxygen therapy for such patients. He might well have gone even further and advocated "aid" to respiration. On page 58 technique is described for the "single-dose" administration of vinyl ether for tonsillectomy in children. One might have imagined that such technique and the surgical methods which evoked it might be allowed to fall into desuetude. Reference is made more than once in the text to the value in resuscitation of solutions of glucose administered *per rectum*. The fact that glucose is not absorbed from the rectum was demonstrated by Corkill in 1936: its use merely hinders osmotically the absorption of water.

In reading a work such as the present one needs to recall that it was written largely for the candidate for a senior qualification in anæsthetics. Such a candidate may come from any one of many schools, at which diverse methods are employed. The author has therefore set out to describe, in a single pocket-sized volume, the legion techniques of modern anæsthesia. Whilst this fact confers high utility, it enforces caution upon the reader. Some of the methods described are outdated; others are, to our thinking, unsound. Such vagaries are not necessarily the author's own, but it is not always clear which is his voice and which that of his authority. The reader, presented with a variety of methods for handling a given problem, may lack experience to choose between them. It is then the author's part to supply direction, to clear away undergrowth and to signpost false trails.

The "Synopsis", in its present form, will serve a useful purpose. We may hope that, in the third edition, Dr. Lee will allow his own personality to appear and will give his own evaluation of the techniques which he describes. It is a task for which he appears to have every qualification.

MAJOR SYMPTOMS.

IN the second volume of "Major Symptoms in Clinical Medicine" John Almeyda covers neurological, psychological, dermatological, locomotor and endocrine symptoms.[1] Much care has obviously been taken in the selection of subjects for each chapter. The important presenting symptoms and signs are studied in great detail. The anatomical and physiological aspects of the various symptoms are well covered, then follows a classification of causes, and finally a suggested mode of investigation in cases presenting with such a symptom. It is here that the main weakness of the book reveals itself. It is often little more than a very brief account of ideas which may come into the mind of the clinician as he reads a description of such a condition in a text-book, rather than an account of the way in which a well-trained physician would act if confronted with a case. In his preoccupation with the symptom the writer appears to lose sight of the fact that most physicians take a full history and make a complete physical examination of the patient, not confining themselves to the system to which the presenting symptom is referable. Consequently, in describing the investigation of the condition of a patient presenting with monocular disturbance of vision, the reader is told to consider the history, carry out perimetry, examine the ocular, intraocular retinal and retrobulbar structures, and then, if these procedures have failed to elucidate the condition, examine the central nervous system.

The illustrations are excellent. Some are original, but many are reproduced from standard text-books of medicine and neurology. The arrangement of the book facilitates reference and there is a good index. The pages are numbered consecutively from the first volume.

The section on neurology lends itself to the method of arrangement adopted in this book. The anatomical aspect of each symptom is very well described, with well-chosen diagrams, and the subjects selected, such as abnormal ocular movements, vertigo, deafness, headache, coma, ataxia, frequently present problems in both consulting and examination rooms.

The section on arthritis is brief. The classification given is not a standard one and includes rheumatoid arthritis under the heading of infective non-purulent periarthritis. The diagnosis of rheumatoid arthritis is not well done, no mention being made of the value of observing the distribution of the lesions.

The section on endocrinology is devoted mainly to a discussion of the anatomy and physiology of the endocrine system. The symptoms discussed are disorders of menstruation, sterility, frigidity and passion, and impotence. In each case the discussion is brief and relatively superficial. Other symptoms, such as obesity, abnormalities of pigmentation, gigantism and dwarfism, have been dealt with in other sections of the book and the cross reference only is included under endocrinology.

In general this book represents a novel approach to certain aspects of medicine and is a useful supplement to general reading. The arrangement of the subject matter facilitates reference. It should prove valuable to candidates preparing for examinations for higher medical qualifications.

[1] "Major Symptoms in Clinical Medicine", by John Almeyda, M.R.C.P., D.P.H. (London), M.R.C.S. (England), with a foreword by Sir Adolphe Abrahams, O.B.E., M.A., M.D. (Cambridge), F.R.C.P. (London); Volume II; 1951. London: Henry Kimpton. 10" × 6¼", pp. 344, with 137 illustrations, six in colour. Price: 25s.

DERMATOLOGY.

"THERAPY of Dermatologic Disorders" adds yet another to the many books on skin diseases that emanate from the United States of America.[1] The title is somewhat of a misnomer, because all aspects of skin diseases are described, with the main emphasis, however, on treatment.

An interesting departure from the conventional text-book is the complete absence of illustrations except for four diagrams. These diagrams are intended to be an easy aid to the diagnosis of 52 common diseases of the skin.

The authors have drawn up a table of 52 skin disorders met with in everyday practice and have made an attempt to explain the main points in predominant lesions, areas of predilection, subjective symptoms, differential diagnosis and ætiology. Each complaint is given a number from 1 to 52, and by reference to the four diagrams with a number on the site of the lesion one is able to attempt a diagnosis from the study of the chart; this is acknowledged to be drawn from Saboraud's "Topographic Dermatology". Although of some value, this method of diagnosis does not make the same appeal as good illustrations.

From the therapeutic aspect, the authors in the preface stress the treatments they have found of most use. This is as it should be, but unfortunately the plan is not always adhered to, so that an inexperienced practitioner could take his choice from the many methods offered.

This is almost certainly the most up-to-date book on skin therapy that has yet been published. Nothing of recent development seems to have been missed. The latest treatment is not necessarily always the best.

For *impetigo contagiosa* four ointments containing medicaments ranging from aureomycin to sulphathiazole are recommended before there is any mention of a mercurial prepara-

[1] "Therapy of Dermatologic Disorders: Including a Guide to Diagnosis and a Dermatologic Pharmacopeia", by Samuel M. Peck, B.S., M.D., and George Klein, M.D.; 1951. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9¼" × 6", pp. 384, with four illustrations. Price: 70s.

tion. It is well known that a weak mercurial preparation in this country is cheap and effective.

For obstinate patches of neuro-dermatitis heroic applications, such as carbon dioxide snow or a 75% solution of trichloracetic acid, are suggested with due caution. This is indeed an heroic approach.

No description of radium or X-ray technique is included—rather a pity in such an expensive book.

A really excellent chapter is the "Dermatologic Pharmacopeia", and nurses would be interested in the concluding article, "Removal of Medical Stains".

Although this volume is a mine of dermatological information, it is not recommended for medical students or general practitioners. It should be well received by experienced and junior dermatologists as a stimulating and scientific publication.

MEDICAL AND PHYSICAL DIAGNOSIS.

THE eighth edition of Loewenberg's "Medical and Physical Diagnosis" was published in 1951 as evidence of the continued popularity of this work.[1] The book aims to cover the general field of diagnosis in internal medicine. It includes an analysis of symptoms and general signs as well as methods of examining clinically the various bodily systems and regions. Certain anatomical and physiological principles are enunciated before each system is discussed. In addition, useful sections deal with geriatrics and special physical examination for life insurance and positions in industry. Current laboratory methods are also outlined.

The book contains a vast amount of information, and liberal use of headings and subheadings makes the text easy to follow. In general, the illustrations are fairly good, though black and white pictures are always inadequate to illustrate skin rashes, and some of the line diagrams are confusing.

While one admires an all-round physician who can alone compile such an immense work in an age of specialism, the book shows signs of the author's heavy burden. Lack of specialist consultation is reflected in the complete absence of reference to unipolar leads in the electrocardiographic section. More careful editing is necessary to remove a number of obvious errors: for instance, the lower level of the *manubrium sterni* described at the level of the ninth thoracic vertebra (page 213), pupillary dilatation of "3 to 5 centimeters" (page 960). The text is at times obscure and a number of the definitions are loose. Many will disagree with certain of the author's classifications. For instance, diseases of the breast are considered under the respiratory system, and malingering is held to include simulation of health by those with disease, as well as the reverse. Though the English purist has perhaps learned not to wince at orthodox American spelling, he still writhes at such American slang as the "neuro" who "hollers loud and long" on pages 955-956.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The 1951 Year Book of General Surgery (July, 1950-May, 1951)", edited by Evarts A. Graham, A.B., M.D., with a section on anæsthesia edited by Stuart C. Cullen, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 8" × 5½", pp. 630, with 209 illustrations. Price: $5.00.

One of the "Practical Medicine Series" of year books.

"Diseases of the Ear, Nose and Throat: A Textbook of Clinical and Laboratory Procedures", by Georges Portmann, M.D., translated by Fernand Montreuil, M.D., and Jules G. Waltner, M.D.; 1951. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 7½", pp. 736, with 11 plates in colour and 666 illustrations. Price: £10 15s.

A second edition of a book reviewed in these columns on October 21, 1939.

"Surgical Technique", by Stephen Power, M.D., F.R.C.S.; 1951. London: William Heinemann (Medical Books), Limited. 7½" × 5", pp. 390, with 198 illustrations. Price: 30s.

The author is concerned with "minor technicalities".

"Chronic Bronchitis", by Trevor H. Hewell, M.R.C.P.Ed.; 1951. London: Butterworth and Company (Publishers), Limited. 8½" × 5", pp. 120, with two plates and 26 text figures. Price: 27s. 6d.

The author is a physician attached to a "Geriatric Research Unit" and he lectures in problems of old age at Saint Bartholomew's Hospital, London.

"Tumors of the Skin: Benign and Malignant", by Joseph Jordan Eller, B.S., M.D., and William Douglas Eller, M.D.; Second Edition; 1951. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 698, with 550 illustrations and three plates in colour. Price: £8 1s.

Contains clinical descriptions of the various neoplasms with discussions on therapy based on the experience of the authors and of others who are named.

"Internal Medicine: Its Theory and Practice", by John H. Musser, B.S., M.D., F.A.C.P.; Fifth Edition edited by Michael G. Wohl, M.D., F.A.C.P.; 1951. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 10½" × 7½", pp. 1564, with 236 illustrations and 10 plates in colour. Price: £8 1s. 3d.

The object of the book is "to present in one volume a comprehensive survey of the entire field of internal medicine"; eighty collaborators have been assembled to achieve this end.

"The Hair and Scalp: A Clinical Study (with a Chapter on Hirsuties)", by Agnes Savill, M.A., M.D. (Glasgow), F.R.C.P.I.; Fourth Edition; 1952. London: Edward Arnold and Company. 9" × 6", pp. 328, with 59 illustrations. Price: 25s.

The third edition was published in 1944.

"Nerve Impulse: Transactions of the Second Conference, March 1-2, 1951, New York", edited by David Nachmansohn, M.D.; 1951. New York: Josiah Macy Junior Foundation. 9½" × 6½", pp. 204, with 37 text figures. Price: $3.50.

Consists of five papers and discussions.

"Factors Regulating Blood Pressure: Transactions of the Fifth Conference, February 15 and 16, 1951, New York", edited by Benjamin W. Zweifach and Ephraim Shorr; 1951. New York: Josiah Macy Junior Foundation. 9" × 6", pp. 238, with 49 illustrations. Price: $3.75.

Consisting of eleven papers and discussions.

"The Quiet Art: A Doctor's Anthology", compiled by Robert Coope; 1952. Edinburgh: E. and S. Livingstone, Limited. 7½" × 5", pp. 294, with one illustration. Price: 12s. 6d.

A series of "acceptable words".

"La Maladie Infectieuse: Microbes Pathogènes, Méchanismes et Modalités de L'Infection, Applications au Diagnostic et au Traitement", by V. de Lavergne; 1951. Masson et Cie: Paris. 10" × 7", pp. 374. Price: F.2200.

Intended for the clinician and the bacteriologist.

"How to Improve Your Sexual Relations", by Edwin W. Hirsch, M.D.; 1951. Chicago: Zeco Publishing Company. 7½" × 6", pp. 64. Price: $1.00.

Consists of a series of statements on different aspects of sex and sexual behaviour.

"Adrenal Cortex: Transactions of the Second Conference, November 16-17, 1950, New York", edited by Elaine P. Ralli; 1951. New York: Josiah Macy Junior Foundation. 9½" × 6½", pp. 210, with 68 text figures. Price: $3.00.

Comprises five papers and discussions.

"Conference on Problems of Aging: Transactions of the Thirteenth Conference, February 5-6, 1951, New York", edited by Nathan W. Shock; 1951. New York: Josiah Macy Junior Foundation. 9½" × 6½", pp. 194, with 24 illustrations. Price: $4.00.

Six subjects are dealt with by papers and discussion.

[1] "Medical and Physical Diagnosis: Interpretation of Findings", by Samuel A. Loewenberg, M.D., F.A.C.P.; Eighth Edition; 1951. Philadelphia: F. A. Davis Company. Sydney: Angus and Robertson, Limited. 10½" × 7", pp. 1264, with 717 illustrations, 41 in colour. Price: £7 5s. 3d.

The Medical Journal of Australia

SATURDAY, MARCH 15, 1952.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

CONGRESS AND WHAT A BUSH PHILOSOPHER SAID.

SOME years ago there lived in the backblocks of New South Wales a bushman who was well known and liked in the district where he lived. He was a big man of fifty or a few more summers, strong, vigorous and reliable. Men of the land liked to employ him. He looked like other bushmen—he wore his large felt hat complete with bits of cork on pieces of string hanging from the brim to keep the flies from worrying him; he did not think it necessary to shave every day and he wore bowyangs on his trouser legs in the custom of the day. He was one of the best of his type, honest and direct, and he looked you straight in the eye. He had been educated largely in the school of experience and unlike many about him he tried to keep himself abreast of what was going on in the world. He would unburden himself to a congenial soul. One day he sighed and declared that: "There's lots of things not known what nobody knows nothink about." On going with him into the subject of knowledge and its uses one was told that "You generally can't tell what you don't know till you find out". These sentences may sound crude and amusing, but the man who spoke them was sincere. They may be used to introduce once more the subject of the next Congress to readers of this journal, the prospective members of Congress. Many workers in medicine spend their whole lives trying to discover the things "what nobody knows nothink about". As we all know, discoveries are made from time to time and they have to be assimilated and put in their proper place and perspective. Some of the people who go to congresses have found new facts either from experience or from the written word. They are prepared to tell what they know in and around the congress meeting rooms. Even the man who thinks he does not know, like our bushman, may, when he talks with others at Congress, discover that he really knows more than he thought he did. Also he may be able to enlarge the experience of other people. In this way we see that our bushman in his second epigram was saying something not only quite obvious but also useful.

The eighth session of the Australasian Medical Congress (British Medical Association), the congress of the whole medical profession of Australia, will, as should already be known, be held at Melbourne from August 22 to 29, 1952. Readers may perhaps be tired of our urgings that they join as members of Congress without any further delay, but the value of these gatherings to individual members and to the whole profession of the Commonwealth is so great that repetition is justified. As we have previously explained, the actual work at the Congress will not be done so much in the sections as in the past—arrangements have been made for the holding of many discussions of the round-table type. These will be arranged in the same kind of way as was adopted with the plenary session at the Brisbane Congress. The Executive Committee wishes to avoid any suggestion that sectional meetings are for a few high-brow specialists rather than for any member of Congress who cares to attend. Of the value and enjoyment that attend informal discussions outside the meeting rooms there is no need to write much. Of all the things which members will "find out", not the least will be a glimpse of the experience of some who have been just names to them and of others of whom they have not heard. These discoveries of the personal kind have a lasting value. Accommodation is a difficulty and latecomers will have none but themselves to blame if they find arrangements difficult at the last moment. Application should be made through the local secretary of each State. The list of secretaries is as follows: New South Wales, Dr. Selwyn G. Nelson, 233 Macquarie Street, Sydney; New Zealand, Mr. G. R. Lee, P.O. Box 156, Wellington; Queensland, Dr. W. J. Saxton, c.o. B.M.A. House, 225 Wickham Terrace, Brisbane; South Australia, Dr. R. C. Angove, 163 North Terrace, Adelaide; Tasmania, Dr. A. Millar, 163 Macquarie Street, Hobart; Victoria, Dr. H. G. Hiller, Congress Office, 426 Albert Street, East Melbourne; Western Australia, Dr. S. E. Craig, 260 Saint George's Terrace, Perth.

THE MOCK TURTLE'S ARITHMETIC.

LOVERS of "Alice" will recall how the Mock Turtle told her of his schooling—that he went to school in the sea and that at his school there were extras—French, music and washing—they were always put at the end of the bill. He could not afford these extras and so took only the regular course which included Reeling and Writhing and the four branches of Arithmetic—Ambition, Distraction, Uglification and Derision. He also learned Mystery, ancient and modern, with Seaography as well as Drawling. The drawling-master was an old conger-eel who gave them Drawling, Stretching and Fainting in Coils. The Mock Turtle had not passed through the hands of the Classical-master who taught Laughing and Grief. Alice was very impressed with this tale of lessons, particularly because they were so called for the reason that they became less every day. Most of us are like Alice—we are taught the three R's as we all call them, but there are some who follow the Mock Turtle. Of our three R's many think 'Rithmetic to be the most needed and of its four branches

will give pride of place to addition rather than to subtraction, multiplication or division. Addition seems to be most important to us right through our lives. We come into the world with nothing and soon have things added to us. First come clothes—binder (sometimes), napkin and what not—then a little food for our stomach's sake and, after this, attention in varying quantities, depending on our place in the family, the yearnings, instincts, repressions and exuberances of our elderly relatives and the inquisitiveness of our siblings. Sometimes from the foolishness of our elders attention has added to it cajolery or adulation or both; these additions are among those that become burdens, or at least are not to our advantage. In time we add to our equipment knowledge and experience on which our characters are based. So we go through life with additions material and additions of the spiritual or mental kind. If we acquire an appreciation of the ridiculous we are thrice blessed; if we pile chips on the shoulder or black dogs on the back we add something that at least is not a blessing. We may let our imaginations run riot over the additions possible to us and we see that what is an addition from one point of view may take away something from another—there is a kind of mental metabolism and the catabolic products may take from us more than we acquire. In other words, subtraction is addition in reverse. Multiplication then is addition many times repeated and division reverses the process. We have to do with the grave and the gay, the durable and the fleeting, the beautiful and the hideous, the bonny and the gaunt, the wise and the not-so-wise. And so it goes. At the end of our lives we may ask what does life add up to —addition again, as always.

But what shall we say of Alice and the Mock Turtle and of the arithmetic discussed by them? Arithmetic must surely be as important to them as to ordinary folk—and some of these like to follow them in their foolishness. Ambition we all know though it may be that we do not call it by its name. Every time we try to do better than well we have ambition and at times everyone does that, except perhaps those who live a cabbage life. Ambition arises in shadowy form from dreams, and the dream-land may be pleasant. It is the same in a prince or a peasant, an archbishop or an artisan, a doctor or those whom he doctors. The way in which men seek to gratify their ambition will depend on their education, their habits and their spiritual beliefs. Ambition may be praiseworthy and lawful; it may be quite the opposite. We recall Shakespeare's words about the "sterner stuff" of ambition and also what he wrote about "vaulting ambition which o'erleaps itself" so that we may be reminded of what has been said about ambition in relation to pride and greed. In the life-time of most of us the worst kind of ambition has been seen to "o'erleap itself". Ambition may be looked on as permissible, if not laudable, if those who try to gratify it, to make their dreams come true, do not batten on the credulities and miseries of their fellow men. Of the Mock Turtle's other branches of arithmetic we may say that distraction is analogous to subtraction because it will (if we may use the phrase) put ambition into reverse. Uglification can well describe vaulting, excessive, selfish ambition, which in reverse becomes derision, and so the Mock Turtle is vindicated.

We cannot leave this superficial and possibly unnecessary discussion without wondering what the Mock Turtle would have done to medicine had he chosen to dabble with it. He would certainly have talked of infernal medicine, and possibly of guinea-collegey, of "awful Phœnix" or of the pretty ant-tricks of children. Some of his relatives would surely have had an ulster in the stomach, or a guitar in the nose, to say nothing of very close veins or an harmonium in the chest, or even a vicious enema; they may even have taken "cremoniated stincture of Queen Anne". To hear the truth about this we shall probably have to wait for the coming of a second Lewis Carroll.

Current Comment.

BRONCHIAL ASTHMA AND THE ADRENAL CORTEX.

The prominence of the adrenal gland in medical research and practice has directed attention once more to observations made some considerable time ago. Klaus A. J. Järvinen, in a study of the relations between bronchial asthma and the adrenal cortex, reminds us that adrenaline was first used by subcutaneous injection for asthma in 1903, and adrenocortical extract for the same purpose thirty years later.[1] He also recalls the work of the late G. W. Bray, well remembered by his Australian colleagues, who pointed out that the frequent low blood pressure and the tendency to hypoglycæmia in asthma suggested adrenal insufficiency. The characteristic blood sugar tolerance curves in Addison's disease and the sensitiveness of the adrenalectomized animal to anaphylactic shock indicated the possibility of adrenal damage in allergic disease. More recently other workers have obtained evidence of hypofunction of the adrenal cortex, in particular of the mineralocorticoids and glucocorticoids. Järvinen has attempted to show that observations on body weight and blood pressure might point to an adreno-cortical insufficiency, and investigated also the effects of restriction of sodium chloride and exposure to a low barometric pressure. The investigation involved the observation of 625 patients who had suffered from attacks of bronchial asthma for an average period of over ten years. Blood pressure measurements were made during the intervals between attacks, and these were analysed according to age and the duration of the asthma. Within the different age groups the systolic and diastolic readings were of normal level or slightly higher, and the duration of the asthmatic attacks had no demonstrable effect on the pressure. Body weight is usually reduced below the average figure in the subjects of severe or continued attacks, since it is difficult for the patients to take adequate amounts of food. In estimating any possible effect of asthma on weight, Järvinen took into consideration stature, age and sex. On the whole the weight of patients subject to asthma was lower than average for age and build, especially in the male sex, but the difference was not significant. The duration of attacks had no discernible influence on weight. It was observed that 17% of the patients were corpulent.

The sensitiveness of patients with Addison's disease to salt lack is known; not only does the patient with the established disease require large amounts of salt, but observation of the effect of a diet with a low sodium content has been used as a test for adrenal insufficiency. Therefore Järvinen tried the effect of restricting the amount of sodium chloride in the diet of persons suffering from asthma. Other investigators have not found that the sodium content in the blood serum was reduced in the subjects of asthma, or any other evidence of exacerbation of the attacks when the sodium in the diet was restricted, rather the reverse. Järvinen made tests on 22 patients;

[1] *Acta medica Scandinavica*, Volume CXL, Fasciculus VI.

some had a strict salt-free diet, others had very little salt contained in their food and had no added salt. The results gave no indication of any connexion between salt deficiency and attacks of asthma, for none of the patients showed any variation of symptoms during the test period. A small series of three patients was subjected to low atmospheric pressure in a low pressure chamber used for testing flying personnel. They were kept at a pressure corresponding to an altitude of 3500 to 4000 metres for a period of forty to forty-five minutes, and symptoms of a constant type were observed. At first the subjects were somewhat distressed, but their breathing showed no change, and after ten or fifteen minutes they felt relieved, and produced by coughing some whitish sputum. Râles previously heard in the chest disappeared, and an increase in the number of leucocytes was found, though no constant changes in the numbers of eosinophile cells were observed. The good tolerance of low barometric pressures was taken as evidence of an ability of the adrenal cortex to secrete both mineralocorticoids and glucocorticoids, and therefore no evidence was adduced of insufficiency of the gland. In discussing the results, Järvinen remarks that the symptoms of bronchial asthma are relieved by various forms of stress, but he considers that the causative agent producing the relief so gained is adrenaline, possibly by reason of its antagonism to histamine.

THE EXO-ERYTHROCYTE PHASE OF THE MALARIA PARASITE.

THE popular eye tends to see the medical research worker in an air-conditioned chrome-and-tile laboratory, surrounded by every advantage of equipment and staff, steadily and relentlessly conquering the scourges of mankind. Frustrations and primitive resources are relegated to past history; so that it seems a far cry to the tragic events of half a century ago, when Carroll, Lazear and their soldier volunteers in Cuba tussled successfully with yellow fever at the cost of Lazear's life, or to the many difficulties of Ronald Ross at Secunderabad, when in the torrid summer of 1897 he demonstrated that the anopheles mosquito was the carrier of the malaria parasite. It is only too true, however, that the field worker of today, and the laboratory worker, too, often have much to contend with. In such circumstances a sense of humour is an important part of a research worker's natural equipment and may help him to make light of very real difficulties. It is evidently possessed by H. E. Shortt,[1] who has recently sketched the story of the part that he and his colleagues have played in the identification of the "tissue phases" of the malaria parasite. In 1920 Anschutz observed development of a plasmodium in the cerebral capillaries of a Javan sparrow. In 1937 James and Tate gave a full description of the exo-erythrocytic phase of *Plasmodium gallinaceum* in the brain of the chicken. This excited Sinton and Shortt (the present author) to attempts to discover whether such stages existed in mammalian malaria. They selected *P. cynomolgi* and the monkey *Macaca mulatta*. The intervention of war delayed the research. After the war several fruitless attempts were made. Then in 1947 Shortt and his colleagues determined "to do one crucial experiment in which so many sporozoites would be introduced into a monkey that discovery of the pre-erythrocytic stage should be as certain as anything could be". They collected large numbers of *Anopheles maculipennis* from the Essex coast. At one period of the collection one of the assistants slipped into a creek and began to sink in the mud. "As he had the mosquitoes it was important to rescue him because although his loss might have delayed the experiment, the mosquitoes were absolutely essential." He was rescued at the cost of his boots, which belonged to the school. Eventually 500 infected mosquitoes fed on a chosen monkey, and a suspension of 569 mosquitoes ground in a mortar was injected into the same animal. Seven days later the monkey was killed. Many tissue samples were studied without success. The liver was one of the last tissues searched, and it was not until twenty-one days after the autopsy that sections of it were stained. Here the organism was found. The result of this experiment was confirmed a number of times afterwards. The next step was to repeat the experiment in the case of the human parasite *P. vivax*. A volunteer was obtained from the Bucks County Mental Hospital. He was inoculated with *P. vivax* by 2010 mosquitoes over a period of two days, and the salivary glands of 200 infected mosquitoes were injected into one of his veins. Seven days later a piece of liver was removed by open operation. The tissue was cut into small pieces and placed in suitable fixative solutions. Shortt felt that he could not bear to wait the ordinary period of two or three days for the processing of the material. He therefore placed a piece of tissue into a solution that would fix it in half an hour and would overfix it if left much longer. He proposed to transfer it to the washing solution in the train on his way back to London. But the carriage was crowded. He had to wait until he reached Waterloo station, where he made the transfer in a public lavatory "at the cost of one penny". He cut and stained the sections the same night, and eventually found the parasite in its pre-erythrocytic phase, "unmistakable and almost identical with that of *P. cynomolgi*". The next step was to conduct a similar experiment with *P. falciparum*. A human volunteer had to be used from the beginning, because there was no readily available monkey parasite suitable for a pilot experiment. Difficulty was experienced in obtaining a suitable strain of the parasite. The West African strain was not suitable, because *Anopheles maculipennis* was an indifferent host. The Indian strain was too dangerous. Efforts were made to obtain a suitable strain from Greece; but this was difficult, because of the success of an anti-malaria campaign and because of the frequency of mixed infection. "Finally, Dr. Foy, in search of our material, got isolated by brigands in a village and things became altogether too difficult." Eventually a suitable strain was obtained from Rumania. A Mr. Howard, of the Department of Civil Aviation, volunteered for the experiment. But the Treasury at first refused to grant him leave, although, Shortt believes, his own department had no objection, "even if they thought him mad". All efforts failed to move the Treasury until the critical day when the volunteer must be infected by the prepared mosquitoes. The "glands of the mosquitoes were swarming with sporozoites and the mosquitoes had been starved for two days and would die if not fed". Shortt had a chimpanzee in readiness to use at the last moment if necessary. When he had almost given up hope, he received a telephone message that Mr. Howard had been granted the necessary leave, but without pay. Shortt did not learn until some time afterwards that his chief technician had his bag packed in readiness to go to hospital to be inoculated himself if Mr. Howard failed to appear. However, the experiment was duly conducted. The volunteer was operated on 140 hours after the first inoculation. Again Shortt could not wait for the usual processing of the tissues, and he put a small portion through a rapid preparation. However, on this occasion, he gave up the search at 2.30 a.m. He did not find the parasite in its pre-erythrocytic phase until a few hours later. He mentions that he had promised to let Sir Neil Fairley know if he found the parasite during the night. He learnt later that Sir Neil had a very bad night's sleep. He concludes his story with a "dramatic postscript". The volunteer, Mr. Howard, had a history of duodenal ulcer. Some time after the experiment he suffered a relapse, and a duodenal ulcer ruptured. The surgeon who operated said that Mr. Howard's life had probably been saved by the previous operation on his liver. "This had left certain adhesions which had sealed off the perforated area from the rest of the abdominal cavity and prevented a general peritonitis." This was an entirely coincidental result of the liver biopsy, but no doubt Mr. Howard, and perhaps his surgeon, would differ from Shortt and most other people in their assessment of the value of the original experiment.

[1] *Transactions of the Royal Society of Tropical Medicine and Hygiene*, October, 1951.

Abstracts from Medical Literature.

PÆDIATRICS.

Congenital Defects in the Offspring of Pregnant Mice Treated with Cortisone.

F. C. FRASER AND T. D. FAINSTAT (*Pediatrics*, October, 1951) describe the production of congenital defects in the offspring of pregnant mice treated with cortisone. They state that it is already known that a number of environmental agents, when applied to pregnant animals, will cause congenital defects in the offspring. These agents include diet deficiencies (in vitamin *A*, riboflavin, pantothenic acid and folic acid), X-radiation, hypoxia, and injection of trypan blue, nitrogen mustard and possibly desoxycorticosterone acetate. In general, the incidence and type of defect vary according to the agent used, its dosage, the stage of gestation at which it is applied and the genetic constitution of the animal involved. The authors gave cortisone in varying doses to pregnant mice of a number of genetic strains. As a result, intrauterine death or cleft palate was produced in many of the litters. The effects produced varied with the strain of mouse, some being more sensitive than others. The effect ran parallel with the dose of cortisone used, the greater the dose, the greater the effect. The effect varied with the stage of gestation, being more severe early in pregnancy. Work is now being carried out to see whether ACTH has similar effects.

Congenital Megacolon.

ORVAR SWENSON (*Pediatrics*, October, 1951) has published a follow-up report on 82 patients treated surgically by his resection operation for Hirschsprung's disease. He describes the pathology of the disease as an absence of ganglion cells in Auerbach's plexus in the rectosigmoid and rectum; this segmental congenital defect in the nerve supply to the bowel accounts for the absence of peristalsis and produces the narrowed segment of bowel so typical of the disease. Surgical resection of the functionally defective area of bowel relieved these patients. The author states that it is important that the diagnosis be made with accuracy. Conditions that particularly must be excluded are anal stricture, tumours and chronic constipation, which are likely to be associated with fæcal impaction in the rectum. Congenital megacolon, on the other hand, may be associated with fæcal impaction in the sigmoid colon; with it constipation usually dates from birth, abdominal distension is pronounced, and the narrowed segment of recto-sigmoid can be demonstrated by careful opaque enema examination. Diagnosis is difficult in the newborn and young infant because distension of the colon has not yet occurred. Careful preparation for operation is made. For thirty days the child has a low-residue diet, mineral oil by mouth and a daily enema. For the last fourteen days sulphasuxidine is given by mouth to help produce the liquid bacteria-free stool. Bowel irrigation with a tube passed up into the colon is carried out for a day or two before operation, so that distension is completely relieved by the time of operation. Almost invariably the narrowed segment of bowel has been resected by a one-stage, pull-through operation, leaving the anal sphincter intact. Post-operative gastric suction is carried out for one or two days until peristalsis resumes; the taking of fluids and solids is then resumed by mouth. Pre-operative examination of the bladder is carried out because congenital atony of the bladder is common, owing doubtless to some autonomic nerve defect that has affected the bowel. After operation an indwelling catheter is left in the bladder, and after a few days tidal irrigation of the bladder is commenced and continued until the tenth day after operation. Penicillin and "Gantrisin" are both given to help maintain freedom from infection in the operative area and in the bladder. The author has had two deaths, but otherwise the results have been uniformly good. Abdominal distension has disappeared over a period of six to eight months. Anal sphincter function has been normal in all cases. All patients have gained weight, and their general growth has been normal. Diets have been unrestricted, and no mineral oil, laxatives or enemata have been necessary. Barium enema examination as early as the second post-operative month has shown an essentially normal colon.

Transmission of Poliomyelitis Virus.

ALBERT B. SABIN (*Journal of Pediatrics*, November, 1951) discusses the knowledge available concerning the transmission of poliomyelitis virus. He states that human fæces from patients and healthy carriers are the most significant source of virus in nature. The virus, which is occasionally present in the throat of patients and healthy carriers, does not ordinarily spread in significant amounts by droplets from the nose and throat. It is suggested that to obtain convincing evidence on this point further studies should be carried out on the infectivity of naturally expectorated saliva and naturally emitted mouth spray from patients and their families, but it is considered that it is an unlikely source of significant infection. The period of infectivity is not limited to a few days before and a few days after the onset of the illness, but extends over as long after the illness as virus continues to be eliminated in the stool. The period of greatest infectivity is the few days before and the week after the onset of the illness. Approximately 50% of patients still have virus in their fæces three or four weeks after onset. The virus enters the body via the mouth, in contaminated food or drink or fingers. Filth flies can be carriers of virus, and the importance of food or drink, hand carriage or flies varies in different circumstances in any epidemic. In some outbreaks the family contacts of recognized "cases" are of great importance as carriers, but in large epidemics the "cases" and carriers are widely spread throughout the community. By the time the existence of an epidemic is recognized, there are already so many healthy human carriers spreading the virus that neither complete isolation of patients and their associates nor the destruction of the filth fly can be expected to stop the epidemic. This is the real basis for the general agreement that there is no justification for emergency insect control measures in the hope of stopping an epidemic. However, measures designed to combat flies and to minimize contact of flies with food in markets or homes should receive encouragement without any guarantee of effective protection against the infection, for there are many methods of transmission involved in any large outbreak of the disease. Subclinical immunization occurs during non-epidemic years as well as during epidemics, but strains of great virulence are present during the epidemic. Sensible isolation of affected patients, to lessen the opportunity of infecting others who might come into intimate contact with them or whose food they might contaminate, is, of course, wise; but since there is no evidence for the transmission of poliomyelitis by droplet infection, wholesale avoidance of crowds and public gatherings, exclusion of children from schools, churches *et cetera*, and the exclusion of poliomyelitis patients from general hospital wards are not likely to be of any real benefit. On the other hand, during epidemic periods it is wise to wash hands before eating and to keep fingers out of the mouth, to keep flies away from food and to wash thoroughly any that is eaten uncooked, to keep children out of crowded swimming pools and to avoid intimate physical or fomite contact with members of a family in which a case of poliomyelitis has occurred within three weeks, even though the patient has been removed to hospital.

Cœliac Disease.

WILFRID SHELDON AND ANTOINETTE MACMAHON (*Archives of Disease in Childhood*, October, 1951) report a study of glucose absorption in cœliac disease. They confirm the occurrence of the flat blood sugar curve after oral administration of glucose and state that this curve tends to move towards the normal as recovery proceeds. They go on to state that in normal children the blood sugar curve shows a considerably higher rise when glucose is put into the duodenum than when it is given by mouth, but in cœliac disease this is not so, the duodenal glucose curve remaining flat. Barium meal examination studies did not reveal delay in the emptying time of the stomach or in the movement of the meal through the intestine. A segmentation defect in the small intestine was apparent in most of the children with cœliac disease. The rate at which glucose leaves the stomach was studied by repeatedly withdrawing and returning to the stomach a glucose meal, and it was found that, in general, glucose left the stomachs of children with cœliac disease a little faster than it left those of normal children. It seems, therefore, that the rate of passage of the glucose from the stomach to the bowel is not the cause of the flat glucose tolerance curve in cœliac disease, but that the cause lies rather in some defect, probably absorptive, in the bowel.

Control of Pertussis in a Day Nursery.

ANDREW BOGDAN (*The Lancet*, December 29, 1951) describes the successful use of chloramphenicol in the treatment of susceptible contacts in a day-nursery outbreak of pertussis. Striking clinical results were obtained with chloramphenicol therapy begun in the incubation period or early in the preparoxysmal stage. Early bacteriological diagnosis of infection, especially

in the incubation period, proved an invaluable aid to suppressive therapy. The author states that the periods of exclusion from the day nursery were greatly shortened by the rapid elimination of infectivity. This greatly reduced the economic hardships arising from pertussis in the families concerned.

Aureomycin as Prophylaxis Against Ophthalmia Neonatorum.

ARTHUR M. CULLEN AND SAMUEL G. CLARK (*American Journal of Ophthalmology*, June, 1951) have compared silver nitrate and aureomycin in the prophylaxis of *ophthalmia neonatorum*. They state that 442 newborn infants were treated with silver nitrate, while 1000 received aureomycin. In the silver method, the eyes were wiped with sterile swabs moistened with warm water, and two drops of 1% buffered silver nitrate solution were instilled into each eye. Excess was removed with sterile gauze. In the aureomycin series a 0·5% solution of aureomycin borate was used, two drops being instilled into each eye. Of the 422 infants receiving silver nitrate prophylaxis, 115 (26%) developed varying amounts of purulent discharge. None of these received treatment. Of the 1000 infants receiving aureomycin prophylaxis 23 (2·3%) developed purulent discharge, which also cleared without treatment. The natural explanation of the inflammatory reaction occurring after silver nitrate prophylaxis is that, since the superficial epithelial layer is destroyed by the drug, bacterial invasion is likely neither to occur nor to clear so rapidly. In the silver series 50% of cases in which cultures were made yielded organisms, and in the aureomycin series 61% yielded organisms. No infection with gonococcus occurred in either series. The authors express the opinion that the immediate silver reaction is diminished if freshly prepared, nearly neutral silver nitrate is used, and if irrigation with normal saline is practised after the instillation. The eyes of infants are susceptible to pyogenic infection; infection at home would be reduced if the mother was given penicillin or aureomycin to use at home.

ORTHOPÆDIC SURGERY.

Osteoperiosteal Ribbon Grafts in Fractures of the Tibia.

IRVINE M. FLINN (*The Journal of Bone and Joint Surgery*, July, 1951) discusses the use of osteoperiosteal ribbon grafts in eight cases of non-union of the tibia in relatively undisplaced fractures. He states that union occurred in all these cases except one. It was necessary to repeat the procedure in this single failure, and successful union then resulted. Analysis of the failure revealed that the graft did not span the fracture line sufficiently. The author points out that the operative technique is quite simple and gives a full description of it. He believes that there are several highly desirable features of this type of graft. Avoidance of the fracture site, except for the creation of a suitable bed in which to place the graft, means but a minimum of disturbance of the blood supply to that region. Minor operative trauma and early coalescence of the graft make this also a helpful adjunct in the treatment of delayed union. Because of the relatively short operative time and the simplicity of the procedure, it can be used for patients of all age groups. The author considers that osteoperiosteal grafts of the ribbon type coalesce and unite to the shaft in approximately three months' time. By their presence, union is stimulated within the fracture. In six months' time, there is a solid bar of bone across the fracture, and union of the fracture has progressed sufficiently far for weight-bearing without support to be begun. Infection is no contraindication when the graft is placed in a non-involved area. Union will occur in the same manner as in non-infected cases. The smaller the chips in the ribbon graft, the quicker the graft itself unites solidly into a bar of bone. Non-fixation of the graft is important, but proper placing of the graft is essential. It must lie well above and below the fracture site. The placing of one graft so that it extended insufficiently below the fracture site resulted first in partial union and later in non-union.

Treatment of Recurrent Dislocation of the Sterno-Clavicular Joint.

H. JACKSON BURROWS (*The Journal of Bone and Joint Surgery*, May, 1951) suggests that replacement of the costo-clavicular ligament mechanically is an essential part of the operative treatment of recurrent sterno-clavicular dislocation. He states that tenodesis of the subclavius appears to be the simplest and safest way of achieving such replacement. Two cases are described of acquired recurrent forward dislocation of the clavicle treated by tenodesis of the subclavius in addition to capsulorrhaphy of the sterno-clavicular joint. Full function was restored in both cases, and there had been no recurrence at the time of follow up three years and six months after operation in the first case and twelve months after operation in the second. In the latter case the joint had withstood violence that had shattered the clavicle. Full operative details are given.

Trochanteric Fractures and Nail Plate Fixation.

E. MERVYN EVANS (*The Journal of Bone and Joint Surgery*, May, 1951) states that in most cases general direction of a fracture line is obliquely downward and medially to the trochanteric region. The stability or otherwise of the fracture depends on the line of cortical bone on the medial side of the neck and shaft after reduction. When this medial cortical bone is undisplaced or has been restored during reduction, the fracture is stable. When there is a cortical overlap or destruction which cannot be restored, the fracture is unstable, and some degree of *coxa vara* deformity is to be expected. The unstable type of fracture is met with in about 25% of cases, a figure which is found to correspond closely with the incidence of *coxa vara* deformity both in patients treated conservatively and in those treated by operation. The author believes that when internal fixation has been employed in the stable type of fracture immediate weight-bearing may be permitted. He states that in comminuted trochanteric fractures the comminution is caused by the lateral rotation component of the injury, and the fracture must be nailed in the position of lateral rotation to obtain approximation and alignment of the surfaces. He also states that though penetration into the pelvis is usually considered harmless in the Smith-Petersen nailing operation —the hip being in medial rotation—it is seemingly unsafe when the hip is in lateral rotation, for the wire must emerge near the brim of the pelvis, where the pelvic colon lies against the bone unprotected by peritoneum. The author states that the evidence in support of the claim for lower mortality among the patients treated by operation is overwhelming. The operation should be considered an emergency. The age and general condition of the patient have not affected the decision to operate. The author believes that the older and more decrepit the patient, the more urgent is the need for operation, for in these cases speed in the relief of pain and the provision of early mobility are vital. He considers that two minor details have contributed greatly to the low mortality (namely, 10%): firstly, one or two pints of blood are given intravenously as a routine; secondly, much use has been made of tracheo-bronchial suction at the end of the operation and later.

Prognosis in Tuberculosis of the Hip.

J. DOBSON (*The Journal of Bone and Joint Surgery*, May, 1951) has analysed 320 cases of tuberculosis of the hip joint with the late results assessed three or more years after the patients' discharge from hospital. He found that the primary bone focus involved the acetabulum alone in 39·3% of cases, the acetabulum and femoral head in 34·1%, the head of the femur alone in 19·2%, and the femoral neck alone in 7·4%. In 101 cases widespread destruction of the joint had taken place by the time the patient first came under observation. Premature epiphysial fusion round the knee joint of the affected side occurred in 23% of all patients under the age of fifteen years. When hip disease was complicated by multiple foci of active tuberculosis or by secondarily infected abscesses and sinuses, the prognosis was seriously worsened. Significant late deformity occurred in 38·3% of patients discharged with "sound" fibrous ankylosis, and in 60·5% of those with unstable fibrous ankylosis. Of 187 patients observed for more than three years after discharge from hospital, 174 returned to full activity, seven were partly incapacitated and six were totally incapacitated. Analysis of the late results suggests that the prognosis is best when an adequate period of conservative treatment is followed by some form of arthrodesis operation. The author considers that there can be no short cuts in the treatment of skeletal tuberculosis. He states that whereas an extraarticular ilio-femoral arthrodesis after the age of twelve years results in bony ankylosis in a high percentage of cases, below that age it is a failure; and although the present series does not contain any cases of ischio-femoral arthrodesis, he considers that in the light of further experience, this procedure is the operation of choice in the earlier age groups. Only in a few cases has osteotomy alone produced bony ankylosis.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XI.

MINOR CASUALTIES.

THE majority of minor casualties present little difficulty in diagnosis or treatment, and end happily in restoration of full function. A minority do present diagnostic and therapeutic difficulties, and some are potentially serious despite their insignia.

In dealing with minor casualties under-treatment may lead to prolonged or permanent loss of function, and over-treatment or unnecessary treatment to considerable hardships, particularly when the social and economic background of the individual is not taken into consideration.

During the year 1950-1951 at the Brisbane General Hospital 26,386 casualties passed through the traumatic unit of the Orthopædic Department. Of these, 6297 (or 24%) were injuries of the hand. Many of these hand injuries were of a minor nature, but it is in this particular type of injury that adequate treatment is important, for in the hand things easily go wrong and lead to much trouble.

The ensuing remarks on rest and on the primary treatment of wounds concern all types of wounds, but in particular those of the hand. It is for this reason mainly that they have been set down.

On Rest.

A dislocation is comparable to a fracture because in the one there is a broken bone with displacement, in the other ligaments are broken with displacement. After a fracture has been reduced, absolute rest of the parts is maintained until the broken bone is repaired. Similarly, after a dislocation has been reduced the parts should be kept at rest until the broken ligaments are repaired, and it matters not whether the dislocation is of the hip or the finger. In the case of the hip it is customary to immobilize the joint for a period of approximately five weeks; why not the finger joint?

Persistent pain and/or joint instability following a diagnosis of sprain is of fairly frequent occurrence, fingers, ankles and knees being the favoured joints for this distressing complaint.

Since the commonly accepted treatment of a sprain is mobilization, and that of a complete tear of a ligament is immobilization, the diagnosis should be accurate, and accuracy may not be easy in the presence of pain, tenderness and timidity. Diagnostic errors, however, can be minimized by the use of local anæsthesia to abolish the pain-spasm element, and to allow the complete tear to be revealed by clinical or radiological evidence of separation of the articular surfaces on breaking open the joint. In cases of "severe sprain" of the lateral ligament of the ankle joint, if a local anæsthetic solution is injected the integrity of each of the three bands of the ligament can be tested by taking an antero-posterior radiograph of the ankle with the foot held in the three positions of inversion and plantar flexion, inversion and dorsiflexion and inversion in the mid-position. If no tilting of the talus occurs within its mortice, the diagnosis of sprain may be allowed and a more conservative line of treatment adopted, but if tilting is observed complete immobilization for three to five weeks is indicated.

In the case of the knee, separation of the joint surfaces on either side by half an inch or more indicates treatment by immobilization for at least four weeks if not immediate operation. Tears of the collateral ligaments of the knee joint are often associated with rupture of the cruciate ligaments and/or injury to the menisci. It is for this reason that operative treatment is justifiable as a primary measure, and for this reason that some of the patients treated conservatively do not do well and come eventually to the operating table.

If testing under local anæsthesia as described cannot be carried out, and the integrity of any ligament cannot be proved, it would be well to treat all patients with severe sprains by complete immobilization for a period of not less than three to four weeks.

Rest is indicated for skin wounds no less than for injuries of deeper structures, particularly when the involved skin undergoes considerable movement during function, as it does in the neighbourhood of a joint. A laceration crossing the extensor aspect of a metacarpo-phalangeal joint may, if unsplinted, take a considerable time to heal, and the resulting scar may be lumpy, painful and adherent, and accompanied by a significant loss of function.

When to begin movement after tendon repair has been a controversial subject for many years. It is a common but mistaken belief that early and persistent movements will prevent adhesions from forming. Damaged tissues require rest for their normal repair process, and if irritated by movement of any sort they respond immediately by an inflammatory reaction.

A few days after a tendon operation in the finger the tissues are relatively soft and mobile, without redness or swelling, and there is no discomfort. If this finger is persistently exercised either actively or passively within the next week there is an immediate reaction characterized by pain, swelling and stiffness. If movements are continued, function becomes progressively less; and the longer the reaction lasts, the greater is the ultimate adherency.

A simple law should govern the period of rest required by damaged tissues—absolute rest until repair is sufficiently advanced to allow simple function which will not excite an inflammatory reaction, as indicated mainly by persistent pain and swelling.

A rough working guide to the average rest period required by the various tissues is as follows: skin, one week; muscle, two to three weeks; ligaments, three to four weeks; tendons, four to five weeks; bone, five weeks onwards.

On Primary Repair.

When a wound is received, loss of function occurs as a result of structural damage and of the fibrosis which attends the normal repair process. If primary repair is undertaken it should be axiomatic that as a result of it no additional loss of function will occur. Yet it is an undeniable fact that in many cases considerable loss of function does occur as a result of inappropriate primary treatment.

Perhaps one reason for a poor result following primary treatment of wounds is a belief that in order to restore function the continuity of all divided structures must be restored, and the sooner the better. If such a belief is rigidly practised many derelict fingers, hands or limbs will result.

There can be no doubt that primary repair of tissues should be our ultimate goal, but surgery has not yet reached a perfection of technique or management which justifies the attempt in every case. Chemotherapy has reduced the hazards of infection, but without good judgement and carefully planned and executed surgery its value is much lessened. Chemotherapy is not *per se* the open sesame to primary repair, for infection (if the greatest) is not the only factor concerned in the loss of function following operative repair.

Though primary repair may seem a safe and reasonable procedure in a particular case, it may not be the most advisable. Experience has shown that delayed repair of some structures gives an over-all better functional result than does primary repair. On these grounds alone a divided peripheral nerve or tendon within a fibrous sheath is better dealt with as a delayed procedure than as an urgent primary one.

Repair of deep fascial layers is seldom necessary at all.

When a muscle belly is partly or even completely divided its ends may not need to be accurately opposed; posture and time will restore function without resort to many and massive mattress sutures which serve only to increase the existing damage and subsequent scarring by strangulation.

Bone and skin are the two tissues which do require special attention at the primary operation, and of these the skin demands the greatest respect. The ends of a fractured bone must be aligned and the alignment maintained by one means or another (not by internal fixation if an external method will suffice).

Skin must be repaired or replaced as soon as possible. It cannot be over-emphasized that in the skin lies the key to success in the surgery of wounds. Wounds covered by viable skin seldom become infected; wounds left open frequently do. Even in the absence of frank infection, considerable scarring and distortion of tissues attend the healing of an open wound, making subsequent repair much less effective or even impossible. Reconstructive surgery is made immeasurably easier when damaged structures have been covered by healthy skin at the first operation.

The initial treatment of wounds should therefore aim at the prevention of sepsis and the obtaining of the best skin

coverage possible. If in addition a structure can be repaired without a material addition to the risk of sepsis and at the same time a reasonable assurance of good functional result be given, the repair should be carried out.

In regard to lacerations, when tissues are unyielding as they are in the hand, loss of skin, even when minimal, will create difficulty in closing the wound without undue tension, and it is a fundamental principle that skin wounds should be closed without tension. The execution of this principle is desirable in any situation, imperative in unyielding skin. The tension present at the completion of an operation is not the maximum tension to which the tissues will be subjected. Tissue reaction in the succeeding twelve to twenty-four hours adds considerably to the tension. It is folly not to allow for this reactionary increase and irrational to add to its harmful effects by tight or close-set sutures which cut into the skin margins or strangle the inter-stitch area. A small drain inserted for twenty-four hours is a wise safeguard against excessive tension from reactionary exudate or post-operative oozing.

When the skin edges of a ragged laceration are excised, conservatism is indicated lest closure be rendered difficult or dangerous.

Perhaps the wound need not be sutured at all. Many are the simple cuts which have been "painfully" sutured and ended with complications. Samuel Mihles (1764) asks the rhetorical question: "How do you treat a wound without loss of substance?"—and answers: "By careful replacing of the wounded lips, agreeable to the natural uniformity of the part, by my fingers; afterwards by retaining them in that posture by one kind or other, either of the true or of the dry suture, assisted with suitable compress and bandage. . . ." The dry suture referred to was a strip of adhesive leather.

Many simple incised wounds can be closed by "dry suture", or nothing more than a "compress and bandage". Other wounds such as saw cuts bisecting the end of a finger or toe are best held together by strapping rather than suture.

Some of the free margin of a long shallow oblique cut will die in any case; more of it will be lost if it is accurately sutured in position. There will be no dead tissue to harbour organisms if the apical margin of the flap is cut away and the remainder of the flap held firmly in place by a pad and bandage.

When considerable tissue reaction has been evoked or is expected to occur, suturing is contraindicated. The finger tip crushed by a hammer blow bursts open in one or several places, and through these "bursts" the pulp tissue is herniated by internal pressure. The folly of suturing such a wound is rewarded by increased pain, further destruction of tissue, and greater disability. Rapid decompression should be the aim of treatment, and this is best accomplished by a dressing of 20% sodium sulphate over "Vaseline" gauze together with elevation of the limb.

Beware the wound over the metacarpo-phalangeal joint—the joint may be involved, and the patient may hide the truth concerning the mode of injury.

With the fist clenched the metacarpo-phalangeal joint lies approximately one-quarter of an inch distal to the apex of the knuckle, a very vulnerable spot in any type of injury, particularly the punch injury. A knife blade or an in-situ human tooth readily penetrates the skin and the extensor tendon *cum* capsule to enter the joint. When the injury occurs the fist is clenched; when the examination of the injury is made the hand lies flat on the table with the fingers extended. This change of posture causes the wound in the tendon to move proximally, where it lies unseen beneath the intact skin. By reproducing the position in which the hand was injured, the breach in the tendon-capsule combination is displayed and a potential suppurative arthritis can be dealt with.

The suppuration resulting from Vincent's organisms introduced by tooth puncture during a bout of fisticuffs is one of the most serious infections of the hand and requires radical treatment from the outset. The truth concerning the nature of the injury can sometimes be dragged from the patient only by a description of what may happen if such was the case. Enlarging the arthrotomy with thorough irrigation of the joint and leaving the wound open for a safe period before performing a delayed suture is the treatment of choice. Should infection occur and Vincent's organisms be isolated, immediate amputation of the finger is the safest measure.

The Medico-Social Approach.

Minor casualties, like major ones, may be associated in diverse ways with social and economic problems, and these problems should be considered in the management of a particular case.

A man who earns his living by the pick and shovel will not usually be inconvenienced by a mallet finger, unless, of course, he has a hobby or a second means of livelihood which is incompatible with such a condition. Splintage for four or five weeks will impose on him an unnecessary financial burden, and for what is this hardship borne—a 50% chance only of a normal or near-normal finger.

The stenographer with a mallet finger suffers a serious disability, and considerable time and effort must be devoted to her case, in an attempt to obtain a fully functional finger.

A mallet finger requires treatment only when occupational or social conditions demand it, and if the chances of a good result are to be increased beyond 50%, operative repair is necessary. If treatment should fail, or if in the untreated case it is found subsequently that the deformity is causing disability, arthrodesis of the terminal joint by simple excision of the joint and fixation by a single transverse wire loop through drill holes restores the finger to almost full functional capacity in most cases.

As in the case of the mallet finger, the question "What is the best treatment for a detipped finger?" has no direct answer.

Elaborate time-consuming pedicle graft operations in the case of the unintelligent or unskilled labourer are clearly out of place. Amputation at or near the terminal joint is here indicated. Besides returning the individual to his usual occupation in the shortest space of time, many consider the compensatory gain which goes with the anatomical loss preferable to a repaired finger.

The professional spin bowler whose detipped finger was treated by a pedicle graft was most grateful for the resulting somewhat bulky end of his finger which he was able to use with greater spin effect than before.

When a small raw area is concerned, careful attention to dressing is all that is required; when the area is larger, and the indications for amputation or pedicle graft are not present, a split skin graft cut from the forearm is a very simple measure and effective in a high percentage of cases.

In regard to a fractured scaphoid—a scaphoid is broken, a plaster is applied and use of the limb is encouraged. Time drags on and X-ray examination shows no sign of union at three months—everybody is getting a little anxious and still there are no signs of union. At four months the X-ray picture is the same—what then? Are we to continue immobilization? Even if the opinion that union will occur if immobilization is continued long enough was true, is it justifiable to condemn the individual to perhaps a year or more of what may be total incapacity, and with no guarantee of a permanently painless wrist? Are we to recommend an operation of sorts to hasten union and thus commit him to at least another three months of incapacity without guarantee? Or are we to be influenced by social and economic factors and abandon treatment? What is the cause of the delayed union, and what the end result of each of the methods of treatment referred to?

The answer to the second part of this question is that the end result is probably the same in the majority of cases.

It is true that in some cases the factors concerned in delayed or non-union are reversible and all is well, but in others even if union does ultimately occur there is no guarantee that a painful wrist may not develop in the years to come owing to irreversible changes which have occurred in the cartilage. It is also true that many persons with untreated scaphoid fractures and some whose treatment has failed have symptomless wrists for many years.

It would seem that on the evidence available, the radiologically ununited fracture of the scaphoid after four months of adequate immobilization should be left alone, and the individual returned to his normal social and economic life as soon as possible.

De Quervain's Disease, Foreign Bodies, and Acromio-clavicular Subluxation.

De Quervain's Disease.—De Quervain's disease (stenosing tenovaginitis of the thumb tendons at the radial styloid process) is an occupational disease of the housewife. This hapless creature, unprotected by unions and compensation, must often carry on her occupation though considerably disabled by this disease (and others). De Quervain's disease, once established, does not subside with conservative treatment. Rarely in the early cases the condition may improve considerably only to become painful again at a later date. It is therefore unjustifiable to condemn the housewife to

weeks of disability for the sake of a chance that never comes. Operation as soon as the diagnosis is made carries with it a disability time of not more than seven to ten days, it does not require stay in hospital, it does not require general anæsthesia.

Foreign Bodies.—Small metallic foreign bodies, commonly flying chips from the head of a cold chisel or like tool, very seldom give rise to trouble either immediate or remote. Nor do small fragments of needles. They, therefore, do not require removal except in a few instances when they lie in such situations as the pulp of a finger or in contact with a tendon or a joint capsule. To find a small foreign body when deeply imbedded in mobile tissues is extraordinarily difficult besides being unnecessary.

Imbedded splinters of wood on the other hand demand immediate removal by careful dissection in a bloodless field, for they are bacteriologically contaminated. If they are of any appreciable size merely pulling them out is insufficient, the track must be opened and excised or otherwise decontaminated.

Acromio-clavicular Subluxation.—The subluxated acromio-clavicular joint is another condition which might have less care and attention lavished upon it. Trouble seldom follows this lesion in which the coraco-clavicular ligaments are virtually intact. Displacement occurs owing to tearing of the unimportant joint capsule; the important accessory ligaments are not ruptured. Unless a good cosmetic result is desired, treatment beyond rest until the tissue reaction has settled is unnecessary. Even after three to five weeks of treatment by the Robert Jones strapping technique the bump caused by the uplifted outer end of the clavicle is seldom completely reduced, and it is unlikely that the appearance of the original bump will worry many of the football players who are the subjects of most of these injuries.

The Robert Jones strapping technique, unless very carefully applied and constantly observed, is not without risk of skin damage from pressure, or of ulnar nerve lesions from compression of the nerve between the strapping and the medial intermuscular septum.

Summary.

The importance of rest in the treatment of minor casualties is stressed, and exemplified by cases of sprain, laceration, dislocation and tendon injury.

Primary repair of tissues does not necessarily give the best results. Indications for primary or delayed repair in the various tissues are discussed.

The treatment of cuts and lacerations is described. The treatment of mallet finger, delayed union in scaphoid fractures, and detipped fingers is discussed from the medico-social angle.

Short notes are given on the treatment of De Quervain's disease, acromio-clavicular subluxation and foreign bodies.

Acknowledgement.

I wish to acknowledge my thanks to Dr. A. D. Pye for making available to me the statistics of casualties attending the Brisbane General Hospital.

A. R. MURRAY,
Brisbane.

Reference.

Mihles, Samuel (1764), "Elements of Surgery".

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on December 6, 1951, at the Saint George Hospital, Kogarah. The meeting took the form of a number of clinical demonstrations by the members of the honorary medical and surgical staffs of the hospital. Part of this report appeared in the issue of March 8, 1952.

Hirschsprung's Disease Treated by Rectosigmoidectomy.

DR. A. C. THOMAS showed a male child, aged five and a half years, who had been admitted to hospital on April 23, 1950, with the following presenting symptoms: (i) constipation since birth, (ii) colicky abdominal pain of two days' duration, (iii) vomiting of two days' duration, (iv) anorexia present for months, and (v) abdominal distension present for two weeks.

Ever since birth the child had been troubled with constipation, but any abdominal distension which developed had been easily reduced by the use of enemata until the past two weeks, when distension had persisted and increased in spite of enemata, which were no longer well retained. Also over the past few months the child had had anorexia. For the past two days he had complained of colicky abdominal pain and had vomited greenish material. On the child's admission to hospital, his abdomen was said to have been grossly distended and tympanitic; otherwise the child appeared normal. The diagnosis of Hirschsprung's disease was made and confirmed by an X-ray examination on April 29.

The patient was given treatment consisting of (i) regular enemata, (ii) anal dilatation and (iii) the administration of "Prostigmin" and "Carbachol". The distension waned and waxed, but the child was never really free of it. Dr. Thomas was called in consultation on May 16, and it was decided to continue with medical treatment. During the whole of his stay in hospital the child's temperature was within normal limits.

On August 1 the child was given a spinal anæsthetic consisting of 6·5 millilitres of light "Nupercaine" solution, and cutaneous anæsthesia was obtained up to the level of the fourth rib. There were visible peristaltic waves passing across the abdomen, but there was no spontaneous bowel action.

On August 15 the child was given another similar spinal anæsthetic. Once again peristaltic waves were observed, and this time the child had a spontaneous bowel action every day for about one week, after which he reverted to his original condition and regular enemata were required. He was discharged from hospital on October 14.

On November 22 the patient was readmitted to hospital. His general condition was unchanged, and he had had regular bowel actions since his discharge from hospital. His abdomen was still very distended. On November 30 he had a transverse colostomy made. Progress was satisfactory, the colostomy worked well and he was discharged from hospital on December 23.

On August 19, 1951, the child was again admitted to hospital. His colostomy had been working well and he was in good general health. On September 3 the rectum and five inches of sigmoid colon were removed at operation. An indwelling catheter was left in position for four days, and he received penicillin, sulphadiazine and streptomycin. After two days of slight, irregular pyrexia, his condition settled down and he progressed satisfactorily. He was given one litre of blood. The catheter was removed and tidal drainage was instituted on September 7, but was discontinued on September 11, and the child was allowed up. The wound healed well, and there was no constriction at the bowel suture line. He was given a full diet with added protein. The colostomy was closed on September 24 under antibiotic "cover", and after a few days' irregular pyrexia, the child recovered uneventfully, his bowels being open on the fifth day. He was discharged from hospital on November 9.

The resected bowel was examined in the pathology department. It was a piece of bowel six and a half inches long in the fixed state. The proximal dilated portion was three inches long. The distal constricted portion was three and a half inches in length. A sharp demarcation between the dilated and constricted portions was evident on both serosal and mucosal surfaces. A microscopic examination was made of sections of five pieces from the dilated portion, four pieces from the junctional area and four pieces from the constricted portion. Sections of the dilated portion of the bowel showed distinct muscular hypertrophy with the presence of ganglionic cells in the mesenteric plexus of Auerbach. No distinct ganglia were evident in the pieces of junctional area and the constricted portions. Only occasional myelinating fibres could be seen in the dilated portion. They appeared numerous in the junctional and constricted portions.

Polyposis of the Stomach.

Dr. Thomas next showed a female child, aged ten years, who had been admitted to hospital on May 22, 1951, with the following history. She had been well in the morning and had gone to school; but on coming home from school she felt a little sick. She had some soup and toast for tea, and about 7 p.m. she vomited while at the pictures and again on leaving the theatre. The vomitus was dark material. The child had had a similar attack with giddiness, nausea and vomiting of blood in January, 1951; she had been treated by her local doctor with liver injections. For many years

she had suffered from epistaxis, and on those occasions she had vomited blood. The attacks had been very similar to the attack described. The only other illness the child had had was pneumonia at the age of nine months. The child's father had pulmonary tuberculosis and probable carcinoma of the lung.

On examination the child was pale and in no obvious distress. The pulse rate was 140 per minute. There was a mitral systolic murmur. Her blood pressure was 130 millimetres of mercury, systolic, and 80 millimetres, diastolic. No abnormality was detected in the respiratory system. No abdominal tenderness or rigidity was present, and neither the liver nor the spleen was palpable. The tongue was clean and the fauces were clear. There was no evidence of hæmorrhage in the nose or throat. No abnormality was detected in the central nervous system. A half-hourly record of the pulse rate and blood pressure was kept, and sedation with potassium bromide and chloral was instituted.

On June 23 the child did not vomit in the morning, and an X-ray examination of the chest, a Mantoux test and a blood count were ordered. At 6 p.m. on this day the child vomited 16 ounces of blood with some solid food. Her blood pressure fell to 80 millimetres of mercury, systolic, and 50 millimetres, diastolic, and the pulse rate rose to 160 per minute. The child was given morphine and half a litre of blood, and by 1 a.m. on June 24 her blood pressure had risen to 130 millimetres of mercury, systolic, and 80 millimetres, diastolic, and her pulse rate had fallen to 120 per minute. At 8 a.m. on June 24 the child vomited a further 18 ounces of bright blood. At 12 noon a blood transfusion was started. At 4 p.m. her blood pressure was 90 millimetres of mercury, systolic. The child vomited a further four ounces of blood. By 11.30 p.m. she felt better; her systolic blood pressure was 120 millimetres of mercury and her pulse rate was 140 per minute. The blood transfusion was followed by the drip administration of dextrose saline solution.

On June 25 the child felt nauseated and looked pale and ill. She was examined by the honorary medical officer, who ordered more blood (1·5 litres). The liver was said to be enlarged one finger's breadth below the costal margin. The next day the child still felt ill; her blood pressure was 130 millimetres of mercury, systolic, and 80 millimetres, diastolic; her pulse rate was 140 per minute.

On June 28 the child felt and looked much better. There had been no further vomiting. The liver was no longer palpable. Her condition remained good, and the honorary medical officer recommended œsophagoscopy and examination by an ear, nose and throat surgeon. No abnormality was detected in the nose and pharynx. An œsophagoscopic examination was performed on July 6, and no abnormality was detected. An enema was given on July 6 and melæna was found to be present; the patient's condition deteriorated. She was given another 0·5 litre of blood on July 7. The child progressed without further vomiting or melæna. On July 20 an X-ray examination with a barium meal was carried out, and a large irregular mass was seen in the stomach; this was suggested to be a bezoar.

On July 24 the patient was examined by and transferred to the care of Dr. Thomas. Two days later she underwent gastrostomy, and a large papillomatous growth was removed from the posterior surface of the stomach near the lesser curvature. Multiple small papillomata in the stomach were treated by diathermy at operation. After operation the child was given 0·5 litre of blood and 0·5 litre of dextrose saline solution. Gastric aspirations and the intravenous administration of fluids were suspended on the second day after operation, and sterile water was given by mouth. Gradually the diet was increased and the child progressed extremely well. An X-ray examination with a barium enema was carried out, and evidence was obtained of polyposis of the ascending colon and of parts of the caecum. A sigmoidoscopic examination was carried out on August 24, but no polypi were seen. The child was discharged from hospital, well, on August 28.

A microscopic exmination of sections was carried out. The tumours had the structure of adenomata consisting of papillomatous masses of newly formed glands, some of which were cystic. There was pronounced inflammatory infiltration in the supporting stroma and the submucous tissue at the base of the tumours. There was no definite evidence of malignant change, and the over-all picture was that of gastric polyposis. The Mantoux test produced a negative result. X-ray examination of the chest revealed no pulmonary abnormality except for a slight increase in the lung markings in the lower half of the left lung. The appearances roused some suspicion that there might have been a small simple inflammatory lesion at that site. In all, the patient received three litres of blood.

Mesenteric Vascular Occlusion.

DR. T. E. WILSON showed a married woman, aged sixty-three years, who had been admitted to hospital on October 17, 1951, after a severe attack of pain, which was localized at first to the umbilicus. Later the pain moved to the right iliac fossa. Three hours after the onset of the pain she was admitted to hospital. During that interval she had vomited twice. There was no previous history of abdominal pains or indeed of any serious illness. Her bowels were usually regular, but there had been no action on the day of the onset of the pains. On arrival at hospital, the patient was rolling in agony, and was unable to find relief in any position. The tongue was coated and the breath was foul. Tenderness and rigidity were elicited in the right iliac fossa, and tenderness was found in both sides of the pelvis on rectal examination; no blood was present on the examining finger.

The provisional diagnosis was acute appendicitis. At laparotomy (through a McBurney's incision) infarction of a loop of ileum due to thrombosis of a branch of the superior mesenteric artery was found. Two feet of ileum were resected. The administration of penicillin, heparin, and fluids (by the intravenous route) was commenced. On November 23 the patient again developed pain around the umbilicus and in the right iliac fossa. The pain was more severe than during the previous attacks, and she vomited several times. The abdominal signs of tenderness and guarding were similar to those on the previous occasion. Bowel sounds were increased, and no splashing was detected. Again there was no blood on the examining finger after rectal examination. The provisional diagnosis this time was mesenteric vascular occlusion, and treatment with heparin, penicillin, and fluids (by the intravenous route) resulted in apparent cure.

Retroperitoneal Cavernous Hæmangioma.

Dr. Wilson then presented a married woman, aged fifty-eight years, who had been found to have a mobile mass in the right loin at a routine examination. There were no symptoms referable to the mass, which was about two inches in diameter, smooth, firm, not tender, and dull to percussion. The patient's general health was good, and she had never had gall-bladder, kidney or bowel troubles. A plain X-ray examination of the abdomen showed a soft tissue mass in the left loin. A pyelogram showed both kidneys to be functioning normally, but the right ureter was deflected around a spherical mass. Examinations of the urine and blood yielded normal results. At operation on March 21, 1951, the mass was found to be an encapsulated solid tumour in the retroperitoneal tissues. There was no abnormality of the surrounding vessels, and the tumour was removed without difficulty.

The hospital pathologist, Dr. Gatenby, reported that the tumour was an encapsulated cavernous hæmangioma. There was no evidence of malignant change. No family history of angiomata was elicited, and the patient showed no sign of any other tumours.

Functional Results After Radical Mastectomy.

Dr. Wilson presented a married woman, aged sixty years, who had undergone radical mastectomy for a scirrhous carcinoma of the upper and inner quadrant of the right breast on October 17, 1950. A "vertical" incision had been used, and movements were restricted for only two days after the operation. The patient was shown to illustrate the full range of movement and the absence of swelling and other troubles with the arm.

Another patient presented by Dr. Wilson, a married woman, aged thirty-five years, had undergone radical mastectomy for a subareolar scirrhous carcinoma of the left breast on May 23, 1949. The operation had been followed by a therapeutic abortion. She also had a "vertical" incision and was encouraged to use the arm after the first two days. Courses of deep X-ray therapy were given after the operation and again in the last two months because of the appearance of two nodules just lateral to the lower end of the left sterno-mastoid muscle. Again, there was a full range of movement and absence of swelling or any troubles with the arm.

Functional Results After Excision of Fibroadenoma.

Dr. Wilson's next patient, a married woman, aged forty-four years, had noticed a lump in the right breast for three months. It had increased slightly in size during that time. There was no discharge from the nipple, and the lump was not painful. On examination of the patient, there was an

oval mass measuring about three-quarters by half an inch in the upper and outer quadrant of the right breast. It was smooth, firm and not fixed to the skin or muscles. The mass was removed through a periareolar incision. Histological examination showed it to consist of fibrous tissue containing scattered groups of glandular acini and occasional dilated ducts. No cyst was seen, and there was no evidence of malignant change. After two months the scar was practically invisible. Dr. Wilson remarked that the patient since then had undergone scalenotomy for a *scalenus anterior* syndrome with relief.

Perineal Excision of the Rectum in the Aged.

The last patient presented by Dr. Wilson, a man, aged ninety-one years, had been admitted to hospital with the signs and symptoms of a subacute large bowel obstruction for three weeks. He had had a prostatectomy performed by Dr. Pfeiffer some eight years previously. During the past two years he had lost two and a half stone in weight. On examination of the patient, his general condition was found to be good, considering his age and the fact that he had vomited several times in the previous three weeks. His tongue was clean and moist. His abdomen was very distended and tympanitic, and peristaltic waves were visible. Rectal examination revealed a mass which was just palpable at the tip of the finger and was mobile. Sigmoidoscopy revealed a carcinomatous ulcer eighteen centimetres from the anus. A piece of this was taken for examination, which confirmed the diagnosis of adenocarcinoma. On November 14, 1951, a loop colostomy was performed under local anæsthesia, and the neoplasm was palpated in the pelvis about two inches above the pelvic peritoneal floor. After this operation there was no deterioration in his condition, and within two days the abdominal distension subsided and the colostomy acted normally. On November 21, under low spinal anæsthesia, a perineal excision was performed, following which convalescence was very satisfactory. The colostomy was working well, but the wound in the left iliac fossa had not quite healed. The posterior portion of the perineal wound had healed, and there was now very little discharge from the anterior sinus. The patient had been out of bed for up to an hour, but was able to walk only short distances.

(To be continued.)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

EDITORIAL: THE SCARLATINA EPIDEMIC.[1]

[*The Australian*, May 18, 1841.]

We have said that the Medical Report recently put forth, by order of the Governor, appeared to us meagre and unsatisfactory. We have since heard that this comment has excited displeasure in certain quarters. It is well therefore to say a few words further on this matter.

It need not be stated that in requesting the Board to express their official sentiments on this important subject, his Excellency pursued a proper course, and one indeed, which had become imperative upon him. He had received assurance on all hands that scarlatina had made, and was still making, alarming ravages in the town, and it naturally became his duty to require the cooperation of the qualified medical gentlemen, in taking measures of precaution and remedy.

The Report, however, before us, sets out by advancing this extraordinary position:

"The duties of the Medical Board are confined *solely* to the examination and registration of the testimonials of qualification of parties desirous of being enrolled as legally qualified Medical Practitioners."

Indeed! In our simplicity we had thought that the Medical Board had a charge to watch over, second to none in importance, viz:—it had to watch over all the circumstances which concern the health of the community at large. No doubt the Board has likewise to take care, so far to use the confidence which the public reposes in it, as to discourage the practice of any charlatans and medical impostors in the town. But for its members to express their official opinion, that its duties are limited to this latter function, is to say what is neither true nor proper. As though when called upon by the presiding authority, the Board made it a kind of favour (forsooth!) to express any opinion at all with regard to the public health, or the progress of this infectious epidemic.

In a further passage of the Report we find the members stating that the observations they have to offer are "necessarily hurried". This apology for the imperfection of their document, we consider entirely inadmissible—upon what sufficient grounds does the Medical Board say that it is "hurried". Even if his Excellency had given them a day's notice only, a few hours of that day might have sufficed for setting forth any opinion however elaborate, *the materials for which, must, in the nature of things, have been at hand.* The constituent members of the Board had possessed the daily experience for more than two months past of the state of the town as regards disease. They must have long since been deliberately prepared to express their sentiments upon the points bearing upon the requisition of Government. And if, under such circumstances, they were unable, currente calamo, to state these opinions at length, and with due minuteness and perspicuity, this inability argues much for their caution, it is true, but very little for their mental capacity. Of the formal declarations put forth by the Board, the first four are self evident propositions, well known to the public and which it required no Solomon to gravely declare. That sickness prevailed in Sydney, that this sickness consists in scarlatina, that it began in a certain parish in Sydney, that it afterwards extended itself to all other parts of the town and that epidemics have always proved most fatal, when they make their first appearance in particular places, are facts, which as no one ever denied or is likely to deny, need not have been recorded with solemnity in the Government Gazette as, so many proofs of the concentrated wisdom of our Sydney Æsculapii.

The fifth remark comes in the shape of what we have, in our former observations on this matter, termed a very proper recommendation: namely, that inasmuch as the malignity of an epidemic is increased or mitigated by the absence or presence of cleanliness and ventilation, it is increased or mitigated by the absence or presence of cleanliness and ventilation, it is incumbent on Government to have the bye-lanes wharves etc thoroughly cleansed, and if necessary, that the magistrates should be empowered to prosecute all persons, who create, either within or without their houses, nuisances of any kind. And, entirely approving, as we do, of this portion of the Report, we shall look with anxiety to the carrying out by Government of the request therein set forth.

A clause towards the conclusion of the Report refers to the "high rate of remuneration for labour, the reasonable price of food, and the ability of the poor to command a large share of the comforts and necessaries of life". We are reminded in observing these phrases of the old saying, *Ne sutor ultracrepidam*, which being interpreted means "Let every cobbler stick to his last". What the members of the Medical Board, as such, have to do with the rate of wages we know not. This passage looks like a political opinion, thrust endlong under the eye of His Excellency "will he nill he" as the Scotsman says—or *holus bolus* in the classical language of the late Sir William Curtis. The fact is, seriously, that it was in very bad taste for the medical gentlemen to advance any such opinion (right or wrong as it may be). It was their business to confine their answer to the question that was directly asked them, and to have refrained from adverting to any political sentiment whatsoever.

We will observe, lastly, that the Report is deficient in this one great particular, that it offers to the public *no advice* as to the mode of dealing with the present epidemic. It neither specifies the incipient symptoms of the disease, nor does it treat of precautions such as diet and otherwise, or of proper remedies. In short, it abstains entirely from grappling with details, and may be said to consist in no more than a few vague observations, the substance of which was quite known to the public previously. Such being the case we have felt ourselves compelled to speak of this Report as being both meagre and unsatisfactory. It might as well have never been framed at all. The five minutes which may have been spent in its composition are five minutes thrown away, as far as the public welfare is concerned ,and to use a somewhat vulgar but yet pithy and expressive term we cannot but characterize the Report as little more than a piece of Medical HUMBUG.

[1] From the original in the Mitchell Library, Sydney.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

GENERAL REVISION COURSE, 1952.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the annual general revision course will be held in Sydney for two weeks, beginning on April 28, 1952. The course, whilst of a general nature, has been specially designed to give emphasis to the diagnosis and treatment of cancer.

Programme.

The programme will be as follows.

Monday, April 28.—9.15 a.m., registration; 9.45 a.m., opening of course by Sir Charles Bickerton Blackburn, Chancellor of the University; 10.30 a.m., seminar, "New Drugs", Professor H. K. Ward (chairman), Dr. A. W. Morrow, Dr. Edgar Thomson, Dr. J. Loewenthal, Dr. K. Harrison and Dr. F. L. Ritchie. At the Royal Alexandra Hospital for Children, Bridge Road, Camperdown: 2.15 p.m., pædiatric session, Dr. D. G. Hamilton and Dr. T. Y. Nelson.

Tuesday, April 29.—9.15 a.m., "The Cancer Problem in New South Wales", Dr. H. O. Lancaster; 9.30 a.m., "Carcinoma of the Thyroid", Dr. H. R. G. Poate; 10.45 a.m., "Cancer of the Stomach", Dr. N. Wyndham; 11.30 a.m., "Cancer of the Breast", Dr. S. H. Lovell. At Broughton Hall Psychiatric Clinic, Wharf Road, Leichhardt: 2.15 p.m., psychiatry demonstration, Dr. Guy Lawrance and staff.

Wednesday, April 30.—9.15 a.m., "Biopsy in Cancer", Dr. A. H. Tebbutt; 9.30 a.m., "Carcinoma of the Large Bowel and Rectum", Dr. V. M. Coppleson; 10.45 a.m., "Melanoma and Skin Cancer", Dr. K. W. Starr; 11.30 a.m., "Use of Radium and Deep X-Ray Therapy in the Treatment of Malignant Diseases", Dr. H. J. Ham; 2.15 p.m., "Hodgkin's Disease and Allied Diseases", Dr. C. R. Bickerton Blackburn; 3.45 p.m., "The Use and Abuse of Blood Transfusion", Dr. R. J. Walsh; 4.30 p.m., film session.

Thursday, May 1.—9.15 a.m., "Carcinoma of the Uterus", Dr. H. H. Schlink; 10.45 a.m., seminar, "Problems in Gynæcology", to be arranged by Dr. F. A. Maguire; 2.15 p.m., "Dehydration and Water Balance", Dr. F. C. Courtice; 3.45 p.m., "Visceral Neuroses", Dr. C. G. McDonald.

Friday, May 2.—9.15 a.m., "Management of Chronic Nervous Diseases", Dr. Eric Susman; 10.45 a.m., "Early Diagnosis of Intracranial Tumours", Dr. K. B. Noad and Dr. I. Douglas Miller; 11.45 a.m., "Poliomyelitis", Dr. Laurence Hughes; 2.15 p.m., panel discussion, "Problems in General Practice", Dr. H. R. R. Grieve (chairman), Dr. A. C. Thomas, Dr. Gordon Lowe and Dr. R. J. Jackson.

Monday, May 5.—9.15 a.m., seminar, "Respiratory Diseases", Dr. Cotter Harvey (chairman); 9.40 a.m., "Pulmonary Tuberculosis", Dr. Bruce White; 10.5 a.m., "Suppurative Diseases of the Lungs", Dr. H. Maynard Rennie; 11 a.m., "Cancer of the Lung", Dr. H. Windsor; 11.25 a.m., "The Place of Radiology in Chest Diseases", Dr. E. W. Frecker; 2.15 p.m., seminar, "The Diarrhœas", Dr. Bruce Hall (chairman), Dr. Edgar Thomson, Dr. T. C. Backhouse and Dr. Brian Dowd.

Tuesday, May 6.—9.15 a.m., "Coronary Artery Disease", Dr. W. E. Fisher; 10.45 a.m., "Cardiac Arrhythmias", Dr. J. Halliday; 11.30 a.m., "The Place of Surgery in Heart Disease", Dr. Frank Mills and Dr. J. Kempson Maddox.

Wednesday, May 7.—9.15 a.m., "Ear, Nose and Throat Problems in General Practice", Dr. B. Blomfield; 10.45 a.m., "Common Ophthalmological Problems", Dr. Conrad Blakemore; 11.45 a.m., "Dermatology in General Practice", Dr. J. C. Belisario; 2.15 p.m., "Urology in General Practice", Dr. K. Kirkland; 3.45 p.m., seminar, "Anæsthesia in General Practice", Dr. H. Daly (chairman), Dr. W. I. T. Hotten, Dr. S. V. Marshall and Dr. L. T. Shea.

Thursday, May 8.—9.15 a.m., "Treatment of Rheumatoid Arthritis", Dr. Selwyn Nelson; 10.45 a.m., seminar, "Hæmatemesis and Melæna", Dr. A. J. Collins (chairman), Dr. F. Niesche and Dr. Stanley Goulston; 2.15 p.m., "Diabetic Emergencies", Dr. W. W. Ingram; 3.45 p.m., seminar, "Endocrine Problems", Dr. W. Bye (chairman), Dr. C. W. G. Lee and Dr. F. F. Rundle.

Friday, May 9.—9.15 a.m., "Diabetes and Pregnancy", Dr. J. Cobley; 10.45 a.m., seminar, "Hæmorrhage in Obstetrics",

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 16, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	..	..	..	..	..	..	..	..
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	1	..	..	..	..	..	1
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	2(1)	..	..	..	..	..	..	2
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	10(7)	..	..	..	2	..	..	12
Diphtheria	1(1)	5(5)	1(1)	..	1(1	..	..	..	8
Dysentery (Bacillary)	..	2(2)	6(5)	..	..	..	..	..	8
Encephalitis	..	2(1)	..	..	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	..	..	..	9(7	..	..	..	9
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	3	..	..	1	..	..	4
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	1(1)	3(3)	..	1(1)	..	..	..	..	5
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	7(4)	3(1)	12(6)	22(13)	1(1)	..	..	..	45
Puerperal Fever	..	..	2	..	..	..	..	..	2
Rubella	..	1	..	..	1(1)	..	..	..	2
Salmonella Infection	..	..	..	..	..	1	..	..	1
Scarlet Fever	8(6)	7(5)	7(3)	2(2)	1(1)	4(2)	..	1	30
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1(1)	1	..	..	..	..	..	2
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	28(18)	35(26)	12(4)	3(3)	8(5)	5(4)	2	..	93
Typhoid Fever	..	..	1	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	5(1)	..	1(1)	..	..	..	6
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Professor Bruce T. Mayes (chairman), Professor F. J. Browne and Dr. J. Chesterman; 2.15 p.m., "Infectious Diseases", Dr. N. J. Symington; 3.45 p.m., question time, Dr. R. A. R. Green (chairman), Dr. Bruce Williams and Dr. F. L. Ritchie.

Unless otherwise stated, all sessions will be held in the William H. Crago Council Chamber, British Medical Association House, 135 Macquarie Street, Sydney.

The course will be supervised by Dr. G. L. McDonald. Fees for attendance will be as follows: full course, £8 8s.; mornings or afternoons only, £4 4s.; one week only, £4 4s. Early application, enclosing remittance, should be made to the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483. Telegraphic address: "Postgrad Sydney". Candidates may submit in writing questions for the panel discussion and question time. These must be received at the office not later than Friday, April 18, 1952.

Obituary.

EDGAR ASHLEY FALKNER.

WE are indebted to Dr. C. R. R. Huxtable for the following appreciation of the late Dr. Edgar Ashley Falkner, who died on February 13, 1952, at Southport, Queensland.

Dr. Edgar Ashley Falkner was born at Glastonbury, Somerset, England. He studied at the Middlesex Hospital, London, and obtained the degree of doctor of medicine and the Fellowship of the Royal College of Surgeons of England. He practised at Toowoomba, Queensland, from 1897 to 1927. In 1897, as a young visitor from England, he acted for a term as *locum tenens* for the original Dr. Roberts, of Toowoomba, for whom he formed such a warm regard that he decided to accept the subsequent invitation of the older man to stay on in Toowoomba as his assistant in practice. Thus began a career of thirty years which brought to Dr. Falkner the widest esteem and affection throughout that area of the Darling Downs. A man of great height both in the physical and in a cultural sense, he stooped to win the hearts of men and of children, not only by the confidence he inspired, but by a rare charm of voice and kindliness of manner. A true gentleman, without effort he maintained a quiet reserve about his own achievements and charitable acts. He took a great interest in the Toowoomba Grammar School, and in educational matters generally. But it was his own secret that he paid for the medical education of more than one young man, whose parents were not in a financial position to do so. After retiring from practice, Dr. Falkner settled in England and was there throughout the war years. His wife, who was the third daughter of the late Honourable W. H. Walsh, of Brisbane, predeceased him in England. After the war he returned to Australia to live at Southport, where he is survived by his only daughter, Mrs. M. Ponsford.

MICHAEL VEECH.

WE regret to announce the death of Dr. Michael Veech, which occurred on February 21, 1952, at Manly, New South Wales.

ANDREW STUART ROBERTSON.

WE regret to announce the death of Dr. Andrew Stuart Robertson, which occurred on February 23, 1952, at Bairnsdale, Victoria.

FREDERICK JOHN BENNETT.

WE regret to announce the death of Dr. Frederick John Bennett, which occurred on February 25, 1952, at Heidelberg, Victoria.

ROBERT DUNLOP GOLDIE.

WE regret to announce the death of Dr. Robert Dunlop Goldie, which occurred on March 1, 1952, at Wollongong, New South Wales.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Herscovici, Moses Moise, registered in accordance with the *Medical Practitioners Act*, 1938-1951, Section 17 (1) (*c*), 58 Campbell Parade, Bondi Beach.

Barbour, Edmund, M.B., B.S., 1951 (Univ. Sydney), William Street, Bellingen.

Diary for the Month.

MARCH 17.—Victorian Branch, B.M.A.: Finance Subcommittee.
MARCH 19.—Western Australian Branch, B.M.A.: General Meeting.
MARCH 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
MARCH 20.—Victorian Branch, B.M.A.: Executive Committee.
MARCH 25.—New South Wales Branch, B.M.A.: Council Quarterly.
MARCH 26.—Victorian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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